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THE CONSERVATIVE TREATMENT OF APPENDICEAL PERITONITIS

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SO VOLUMINOUS is the literature on appendicitis today that writers almost invariably feel somewhat apologetic about presenting another discussion of the subject. Yet the very fact that this disease is indelible in the minds of the foremost thinkers of the profession indicates its importance as a disease entity and warrants an exhaustive and comprehensive study of the methods of combating this common yet too often fatal disease. We believe that only by persistent statistical study of the results obtained from the employment of any plan of treatment can one hope to evaluate the efficiency of any one method and consequently reduce the mortality rate. Therefore, we propose to present a critical analysis of a series of 303 of our cases of appendicitis treated during the past seven years at the Augustana Hospital. In order to make a comparative study this statistical analysis will be preceded by a résumé of the results A. J. Ochsner obtained from a similar investigation of 1,000 cases at the same hospital. Reference will also be made to a series of 3,771 cases of appendicitis treated by all the staff surgeons at the Augustana Hospital during this same period of time. A discussion of the management of appendiceal peritonitis will ensue with special emphasis on the original Ochsner treatment.

The conservative treatment of acute appendicitis could rarely, if ever, be endorsed by any surgeon. It is universally agreed that the appendix must be removed promptly when it becomes acutely inflamed in order to prevent a spread of the infection beyond the appendiceal walls. If this were done in every case of acute appen-

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dicitis, there would be slight need to discuss the indications for any other treatment. Unfortunately, however, far too many patients with acute appendicitis are first seen by the surgeon after extension of the infection has occurred. It is in this large group of cases with acute appendiceal peritonitis that the conservative treatment is indicated. That there is a definite field for the treatment of appendiceal peritonitis is demonstrated by a consideration of the number of cases of acute appendicitis admitted to the hospital after extension of the infection beyond the appendix has already occurred. Ochsner, Gage, and Garside[1] in a review of a group of consecutive cases of acute appendicitis admitted to the Charity Hospital in New Orleans, La., found that at the time of admission in 78.7 per cent of the cases the infection extended beyond the appendix. Finney's[2] analysis of 1,807 cases of acute appendicitis revealed that in 37.5 per cent there was spreading of the infection upon admission to the hospital.

The original conception of the treatment of acute appendicitis was that of immediate operation regardless of the progress of the inflammation or of the existence of complications. No other treatment was considered feasible until A. J. Ochsner advocated deferred operation in certain indicated cases. The discussion continues between the advocates of the delayed operation and the immediate operation, each group reporting almost equally good results with its plan of management in a selected group of cases (Table I).

The unfavorable results obtained by those who have endeavored to follow the conservative treatment of appendiceal peritonitis can be attributed largely to a misunderstanding of the cardinal principles originally proposed by A. J. Ochsner. Many have interpreted the Ochsner treatment as being the conservative treatment of acute appendicitis. Nothing could be further from Ochsner's original concept It was for acute appendiceal peritonitis that Ochsner first advocated the conservative treatment. This plan of treatment is based upon a correct evaluation of the reaction of the peritoneum to bacterial irritants. Peritonitis begins as a local lesion, which may remain thus or which may spread and become diffuse. At first the peritoneum simply appears pinker and more injected than normal. Then the normal sheen is lost, owing to the deposition of a thin layer of fibrin on the surface, which acquires a frosted or ground-glass appearance. Microscopic examination shows the usual appearance of an inflamed serous membrane. The fluid exudation is excessive. The exudate consists mainly of fibrin threads with a variable number of leucocytes and a few red blood cells. The amount of fibrin deposited depends somewhat upon the infecting organism. When colon bacilli predominate, a thick matting is often deposited on the coils of intestine, binding

TABLE I

RESULTS, DELAYED VS. IMMEDIATE OPERATION

AUTHOR	CASES	DEATHS	MORTALITY PER CENT
Immediate Operation:			
Stanton[3]	113	13	42.00
Doughty[4]	7		28.60
Gardner[5]	122		18.00
Taylor and Schmidt[6]	61		16.40
Easton and Watson[7]	100	12	12.00
Guerry[8]	94	10	10.64
Reid et al.[9]	2,921		6.30
Pattison[10]	1,211	62	5.20
Schullinger[11]	2,653	135	5.08
Potter[12]	200	9	4.50
Hobler[13]	2,260		4.30
King[14]	364		4.20
McLanahan (in children)[15]	179		3.91
Sprague et al.[16]	1,463		2.73
Nuttall[17, 18]	551		2.50
Gile and Bowler[19]	901		1.89
Herrick[20]	217	5	1.84
Bliss and Heaton[21]	2,100	10	0.47
Delayed Operation:			
Taylor and Schmidt[6]	33		15.20
Doughty[4]	7		14.30
Kehl and Rentschler[22]	126	16	12.60
Coller and Potter[23]	85	8	9.40
Gardner[5]	126		8.70
Stanton[3]	83	7	8.50
Holder and Wells[24]	1,031		8.20
Pool[25]	757	45	5.94
Sperling and Myrick[26]	518	29	5.06
Arnheim and Neuhof[27]	332	18	5.40
Kirtley and Daniel[28]	1,000		5.10
Garlock[29]	433	20	4.60
Bunch and Doughty[4]	2,309		2.29
Guerry[8]	135	2	1.40
Ochsner and Percy[30]	1,000	22	2.20
Augustana Hospital	3,771	83	2.20
Authors'	303	4	1.30

them together in a naturally defensive reaction. With a preponderance of streptococci, the fibrin is often very scanty with little or no agglutination of opposing surfaces. The fibrin forms a protective covering and together with the omentum limits the spread of the infection. This protective mechanism may be broken down by various foreign forces resulting in the spread of the infection. One of the chief methods by which bacteria are disseminated over the surface of the peritoneum is by peristaltic movement of the intestine. This peristalsis is excessive following ingestion of food or cathartics. Cessation of peristalsis is a protective phenomenon and occurs concomitantly with the peritoneal changes. With the occurrence of this protective adynamic ileus, there is little danger of the breaking down of adhesions and spreading the infection. Excessive dilatation of the intestine must be prevented, however, if intramural compression of the vessels

is to be avoided. This defensive mechanism may terminate in any one of three ways: Localized infection in the peritoneum may end by resolution without suppuration; adhesions may keep the inflammation strictly limited and localized suppuration occur; or the infection may spread diffusely.

Ochsner reasoned that the natural protective reaction of the peritoneum could be further enhanced by directing treatment toward the inhibition of peristalsis in order to prevent the spread of infection. This physiologically sound argument is the basis of his whole method of treatment. The cardinal principles in this method as employed by Ochsner[31, 32] may be briefly enumerated: absolute prohibition of anything whatsoever by mouth; the employment of gastric lavage to combat distention of the intestine; the administration of retention enemas every three to four hours to carry on nutrition; the injection of morphine to relieve pain (and, Ochsner thought, also to retard peristalsis); and the administration of normal saline solution and glucose by proctoclysis to overcome dehydration. The patient is kept in the Fowler position, and absolute rest is essential. There must be no relaxation whatsoever in this treatment until the patient has been free from pain and fever for at least four days. It should be remembered that Ochsner recommended this regime only as a preparation for operation.

Following the proposal of A. J. Ochsner[33] for the delayed operation, many men became ardent advocates of this plan of management. Important among these advocates is Guerry, who lent considerable impetus to the Ochsner treatment. He[8] reports a series of 94 cases of gangrenous ruptured appendicitis with diffuse peritonitis, which were operated upon immediately. There were 10 deaths, a mortality rate of 10.64 per cent. In a similar series of 135 cases treated by the deferred operative method as advocated by Ochsner, the number of deaths was reduced to 2, a mortality of 1.4 per cent. In Guerry's[34] series of 2,959 consecutive cases, there were 16 deaths (a mortality rate of 0.54 per cent). Guerry attributes this comparatively large reduction in mortality percentage to the employment of the Ochsner method of treatment in the group of cases with appendiceal peritonitis.

Considerable experimental work has been done in an effort to determine the efficacy of the various methods of combating acute appendicitis. A recent report was made by Schmidt and Taylor[35] of research which they undertook in order to compare the conservative and radical methods of treatment. They made a study of 48 dogs in which they produced appendiceal damage of mild, moderate, or severe degree in an attempt to reproduce experimentally the condition of acute appendicitis. These dogs were examined at one-, two-, or three-

month intervals. In this series of 48 dogs, 28 survived, 15 of which were treated conservatively and 13 by immediate operation. The average mortality for the entire series of 48 dogs was 42 per cent. The mortality according to degree of appendiceal damage was 12.5 per cent, mild; 52 per cent, moderate; and 80 per cent, severe damage. In the group with mild damage there was no mortality for the conservatively treated dogs, whereas a mortality of 33.3 per cent was recorded for those dogs operated upon immediately. In the group with moderate damage a mortality of 42 per cent resulted from conservative treatment and of 60 per cent from radical surgical treatment. All dogs treated conservatively in the group with severe damage died, and three-fourths of those treated by operation succumbed. The total mortality following the conservative treatment was 29 per cent and following immediate surgical treatment was 52 per cent. From these results Schmidt and Taylor[35] concluded that the conservative treatment causes a lower mortality than the operative treatment. They also found that most of the deaths were due to generalized peritonitis (65 per cent), and that the occurrence of adhesions in the peritoneal cavity was more than doubled by operative procedure and drainage.

The original report of the Ochsner treatment came from the Augustana Hospital. These statistics at the time of their first publication appeared revolutionary and their authenticity was at first questioned. Subsequent observations have shown, however, that the early statistics reported by Ochsner could not be considered as unusual, and, after more experience with the plan of management, Ochsner definitely improved on his first results.

There have been no reports in recent years from the Augustana Hospital indicating the present status of the treatment that Ochsner originally outlined. With the refinement in treatment now available, better results should be expected and some modification is justifiable. Advance in therapy should be credited to the Wangensteen continuous duodenal suction and to the availability of more satisfactory intravenous solutions. These measures were not used by Ochsner. He did not give fluids intravenously or subcutaneously but placed much dependence on nutrient enemas. Consequently, excessive dehydration not infrequently existed. This has been more definitely explained since McNealy and Willems[36] and Ebeling[37] have demonstrated the futility of depending upon absorption from the rectum of any fluids other than tap water. We have also found that duodenal suction is preferable to intermittent gastric lavage from the standpoints both of efficiency in decompressing the upper gastrointestinal tract and of convenience and comfort of the patient. With minor exceptions, however, the plan of management, as outlined by Ochsner, has been followed by us and other staff surgeons at the Augustana Hospital.

Ochsner[30] made a study of 1,000 consecutive cases of appendicitis which were operated upon at the Augustana Hospital during the thirty-three months from July 1, 1901, to April 1, 1904. The total mortality for the series was 2.2 per cent. The mortality rate, according to classification, is more enlightening and is shown in Table II. Of these 255 patients in the second group, 200 were operated upon immediately after entering the hospital and 55 received the delayed treatment. In Group 3, 21 had immediate operations and 34 were treated by the delayed method. There were 39 immediate operations in Group 4 and 78 received the Ochsner treatment. All of the 33 cases with diffuse peritonitis received the deferred operative treatment. An analysis of the 22 deaths as given by the author reveals some interesting facts: Ochsner believed that all 3 deaths in the first group could have been avoided had the operation been confined to the removal of the diseased appendix. All 5 of the second group were operated upon immediately, and the author expressed the opinion that had they received the delayed treatment 3 and possibly 4 of these fatal cases could have had favorable terminations. Only 1 of the 4 deaths in Group 4 was avoidable, and that was a case in which the appendix was removed when only drainage of an abscess should have been undertaken. The other 3 deaths were due to "murderous use of cathartics," "probable thrombus of the external iliac vein," and "probable infarct due to a thrombus."

TABLE II

MORTALITY IN OCHSNER SERIES ACCORDING TO CLASSIFICATION

TYPE OF CASE	CASES	DEATHS	%
1. Chronic appendicitis or interval operation	540	3	0.5
2. Acute appendicitis without perforation	255	5	1.9
3. Acute appendicitis perforated or gangrenous with abscess	55	0	0.0
4. Acute appendicitis perforated with abscess	117	4	3.4
5. Acute appendicitis with diffuse peritonitis	33	10	30.0

It should be noted here that Ochsner realized the limitations of this method of treatment, as he did not advocate its use in children or in the aged. He observed that old people do not seem to tolerate confinement in bed for any length of time. He felt that the chief reasons for restricting the employment of this method in children were the difficulty of following such a rigid treatment and the lack of sufficient development of the omentum to act as a protection.

The treatment of appendicitis in children requires a slightly different procedure. Most authors[38-41] have agreed that the disease is expressed

in a more serious form during childhood. Gangrene of the appendix is observed much earlier in children (and in the aged) and, because of the underdevelopment of the omentum, little localization occurs and diffuse contamination of the peritoneum rapidly ensues. A factor which increases the seriousness of the condition is the difficulty with which a diagnosis is made due to the lack of cooperation from the patients and the "typical" atypical character of the disease in the very young. Cathartics, which so often unwisely are given to children with vague abdominal symptoms, serve to aggravate an already perniciously grave condition. Many authors[5, 15, 27, 30, 39, 42-45] advocate immediate operation in children with acute appendicitis regardless of the stage of the disease, although Coller and Potter[23] express the opinion that the delayed method should be employed in children with peritonitis because they feel that any treatment would be essentially the same. Ochsner[46] believes that the conservative treatment would save many lives if employed in those cases with a beginning localization, regardless of age.

During the past seven years, we have seen 29 cases of acute appendicitis in children under the age of 13 years. Immediate operation was always done if the inflammatory process was confined to the appendix or had become definitely localized. In our series there were 18 cases operated upon immediately. If we believed that localization was beginning after the infection had extended beyond the appendix, conservative treatment was instituted whenever possible. A prolonged period of conservative treatment has been, in our experience, unsatisfactory. In these cases of children with acute appendicitis with perforation the average interval before operation was performed was 3.8 days, which is, of course, much less than the period of conservative treatment in adults. In children who are sufficiently cooperative to allow the use of intravenous infusions and duodenal suction conservative treatment can be employed satisfactorily, and in our opinion it should be used whenever possible in the presence of diffuse appendiceal peritonitis.

Deaver,[47] an enthusiastic advocate of Ochsner's method, preferred to refer to this treatment as "regulation," by which he meant strict anatomic and physiologic rest. His outline of treatment deviated only slightly from Ochsner's original plan. He advocated rest of the body by placing the patient in the Fowler position, either with the use of pillows or preferably with the aid of mechanical beds. Small doses of morphine for pain, stomach lavage, nothing by mouth, and employment of the Murphy drip contribute to rest of the alimentary canal. He also approved of ice applied to the abdomen to benumb the nerves of the walls of the abdomen. This treatment, however, was reserved for diffused peritonitis. In early cases of acute appendicitis before

the onset of peritonitis, immediate operation should be performed.[48] Deaver[49] pointed out that three signals which demand immediate operation are chill, abatement of pain, and drop in temperature.

A brief mention of the results obtained by Deaver and Magoun[50] in 5,488 appendectomies performed at the Lankenau Hospital of Philadelphia for the twenty-year period from 1900 to 1920 will be indicative of the effectiveness of this plan of treatment. There were 327 deaths, or a mortality of 5 per cent, in the entire series of cases. A detailed analysis of the mortality for five-year periods is further evidence of the value of the deferred method. The average mortality for the first five-year period from 1900 to 1905 was 10.5 per cent. All of these cases were operated upon immediately. From 1906 to 1909 the mortality was reduced to 5.6 per cent. Some of these cases were treated expectantly. From 1910 to 1920, when all spreading peritonitis cases were treated by the "regulation" method, the mortality was reduced to approximately 4 per cent. Keyes[51] reported a mortality rate of 7.3 per cent in 315 cases of perforated appendicitis with abscess formation; whereas in 98 cases of ruptured appendicitis with diffuse peritonitis the mortality rate was 27.5 per cent. Potter and Coller[52] report that when immediate operation was done in cases with appendiceal peritonitis the mortality rate was 52 per cent, and 21 per cent in those not actually moribund on admission. By employing the conservative treatment, the mortality in cases with peritonitis has been reduced to 8.6 per cent and to 5.6 per cent in cases with abscess. These statistics, together with those reported by Guerry, show a decided reduction in mortality with the advent of the Ochsner method of treatment.

In contradistinction to the enthusiasts[53-55] of the delayed operation many surgeons favor immediate operation regardless of the progress of the spread of infection or the condition of the patient. At operation the infected region is walled off from the rest of the peritoneal cavity by suitable placing of moist packs, and the appendix is removed. Drainage is instituted whenever it seems desirable. Among the advocates of the immediate appendectomy may be listed Herrick,[20] Nuttall,[17, 18] Reid and co-workers,[9] Gile and Bowler,[19] Hobler,[13] King,[14] etc. They report mortality rates from 0.47 per cent to 42 per cent (Table I).

During the period from October, 1931, to October, 1938, we have treated 303 cases of appendicitis. Of this number, 107 were classified as chronic appendicitis. In some of these 107 cases other abdominal organs were the site of pathologic changes necessitating surgical treatment, but in all of these cases the appendix was chronically inflamed and definitely diseased. No mention is made in this series of those cases in which the appendix was removed incidently during the course

of abdominal operation for other conditions. In these cases, classified as chronic appendicitis, the appendix was the major surgical indication for the operation. In this series of chronic appendicitis there were no deaths.

There were 143 cases of acute appendicitis which we treated by immediate operation. The average interval between the onset of symptoms and operation was thirty-eight hours. It was our opinion that deferred operation was not indicated in these cases. It is unfortunate that many surgeons have established a rule for employing conservative treatment based on the duration of appendicitis. This is indeed a mistake. Every case of acute appendicitis presents a different problem, and it is impossible to formulate a plan of treatment based solely on the number of hours since the onset of the attack. Some of these 143 cases had been ill with appendicitis for four or five days. In many of these cases the inflammatory process in the appendix had progressed to the gangrenous stage, and in some there was free fluid in the peritoneal cavity. We have always performed immediate operation in those cases in which the appendix has only recently ruptured, and because no attempt at localization preceded or followed the perforation the appendix continues to contaminate the peritoneal cavity. In these cases there are no adhesions and consequently no danger of further disseminating the process by operation. Drainage was employed in only 5 cases in this group of 143. It is, of course, obvious that drainage need not be used in those cases in which the infection is limited to the appendix. The impossibility of draining the peritoneal cavity per se has been repeatedly demonstrated. In the many instances in which free exudate was encountered in the peritoneal cavity no attempt was made to establish drainage. The peritoneum, as has been said above, is able to withstand bacterial trauma very well indeed. In most cases, after removal of the appendix, the exudate is absorbed and spontaneous resolution occurs. In only 1 case which was closed without drainage, did a residual abscess occur. This abscess became localized in the cul-de-sac of Douglas and was easily drained through the rectum. We have noted, however, that in these cases with free peritoneal exudate a subcutaneous abscess may develop in the wound even though we make every effort to prevent contamination of the abdominal wall. For this reason a small rubber tube drain is placed in the wound just down to the fascia and is removed after two or three days.

The question of drainage has probably aroused as much interest as the discussion on the treatment of appendicitis. From a brief survey of the literature,[56-60] the pendulum appears to have swung from "when in doubt, drain," to "when in doubt, don't drain." Willis and Mora[61] briefly reviewed the opinions of earlier writers on drain-

age and found that drainage seemed to have no effect on the mortality. From a study of 100 cases with drainage following operation for ruptured and gangrenous appendicitis, they concluded that drainage was unnecessary, as it did not subtract from the mortality but did add to the morbidity.

A history of catharsis was obtained in 20 cases. This history always suggests the possibility of early extension of the inflammatory process beyond the appendix. Therefore, in cases with a history of having taken cathartics, special attention is directed toward differentiating localizing peritonitis from generalized peritoneal involvement. No attempt is made here to record the many variations in the types of symptoms exhibited by these patients, because it would seem that nothing very beneficial could be obtained by reiterating these various trends of symptoms. The average duration of hospitalization postoperatively in these cases was nine days. There was 1 death in this series, an analysis of which follows:

CASE 1.—H. E. S., white male, aged 48 years, first seen at 9:30 P.M. upon admission to the hospital July 4, 1937, when he complained of pain and tenderness in the right lower abdominal quadrant. Nineteen hours previously he had first noted acute diffuse abdominal pain which he attributed to dietary indiscretion. About an hour after onset of pain he had a bowel movement, which he felt partially relieved his discomfort. He ate some breakfast with the result that there was recurrrence of abdominal pain. He ate nothing during the remainder of the day and by evening had noticed that the pain had become localized in the right lower quadrant. He sought the advice of a physician who sent him immediately to the hospital. Upon admission, diagnosis of acute appendicitis was made, and immediate operation was performed. The appendix, which was found lateral to the cecum and adherent to the lateral abdominal wall, was purplish red in color and markedly swollen. It was sharply angulated in its mid-third by an adhesive band, and at this point gangrene was found. Considerable fluid exudate was present as well as plastic exudate over the appendix and cecum. Following appendectomy two cigarette Penrose drains were inserted. The next day his temperature rose to 103.4° but immediately fell to 100°. On the fourth day it dropped to 99.4° and on the fifth to 99°. Drainage through the Penrose drains was moderate, there was no abdominal distention, and by the fourth day there was a normal bowel movement and passage of considerable flatus. Early in the morning of the fifth postoperative day, while the patient was sleeping quietly, he awakened suddenly and vomited 100 c.c. of dark blood. Within a few minutes the patient was cold and clammy and the pulse rate, which was almost imperceptible, increased to 144. Immediately the patient was given a blood transfusion of 600 c.c. of whole blood. A Levine tube was passed and 650 c.c. of bloody fluid were aspirated. Dark red fluid continued to drain from the Levine tube constantly. Coagulin was administered, 50 per cent glucose was given through the Levine tube, and preparation was being made for another transfusion, but the patient died eight hours after the onset of hemorrhage. Permission for autopsy could not be obtained.

Comment.—We had previously treated this patient for a duodenal ulcer. It seems most plausible to consider that the few days of postoperative starvation served to accentuate the gastric hyperacidity and become a factor in causation of this fatal hemorrhage from the duodenal

ulcer. As concerns the postoperative condition with relation to the appendicitis, it seemed that the patient's progress was satisfactory.

During this same seven-year period we have treated 52 cases of complicated acute appendicitis by the deferred operative method. The average interval between the onset of symptoms and operation was 6.7 days. In all of these cases we considered the conservative treatment as outlined by Ochsner the method of choice, because it insured the patient the best prognosis as to mortality and morbidity. In only 1 case was the period of conservative treatment curtailed and operation performed in an attempt to prevent the spread of a partially walled-off appendiceal abscess. The patient's condition seemed to be growing rapidly worse and it was hoped that extraperitoneal drainage might be of some benefit. Details of this case follow:

CASE 2.—H. A., white male, 44 years of age, carpenter by trade, first examined in his home May 24, 1938, after having been acutely ill for seven days. Onset of illness was characterized by acute diffuse abdominal pain which the patient attempted to relieve by drinking a large quantity of ice water. This served to increase the pain, and in a further attempt to obtain relief he took several cathartics, including citrate of magnesia, castor oil, and Pluto water. Pain became continuously worse, and by the end of twelve hours became localized in the right lower quadrant. At this time the patient consulted a chiropractor who gave him several "adjustments." On May 20, 1938, three days after onset, the patient's condition seemed worse. He was unable to eat because of vomiting and suffered from abdominal distention. Chiropractic treatments were continued and catharsis was again employed. At the time of our first examination the patient appeared desperately ill. There was generalized abdominal tenderness with definitely more pain upon pressure over the right lower quadrant, where an indefinite mass could be palpated. Definite abdominal distention was present, with no evidence of peristalsis, and marked dehydration was shown by dry skin, parched lips and a high fever (103°). The patient was not able to retain anything by mouth and was restless and slightly delirious. He was admitted immediately to the hospital, and routine treatment for generalized peritonitis was instituted. Because of apparent slight improvement on the following day, it was hoped that localization would occur sufficiently to make operation safe within a few days. On this, his second hospital day, his temperature dropped to 102.4°. Large amounts of fluid were supplied intravenously, and with the reduction of dehydration the patient was considerably more comfortable. On the following day (third hospital day), however, his condition grew worse; abdominal distention increased in spite of continuous duodenal suction and his temperature again rose to 103.4°. It was thought that drainage of the localizing abscess, if accomplished without shock, might be beneficial. A McBurney incision was made close to the right iliac crest, the peritoneum lateral to the abscess was exposed, and eight Penrose cigarette drains were introduced into the lateral side of the abscess. An enormous amount of pus drained away immediately, and the previous treatment was continued. The patient's condition did not improve, the temperature continued to rise, and on the fourth day following institution of drainage death occurred.

At autopsy almost the entire peritoneal cavity was found to be filled with foul-smelling creamy pus. There were unsuccessful attempts at walling off this pus in several areas. The largest loculated collection was in the right lower quadrant in the appendiceal region. This cavity was being partially emptied by the Penrose

drains. In the center of this large abscess the appendix was found. It was completely gangrenous and had partially sloughed away. A considerable amount of pus was collected in the subphrenic and subhepatic areas. All the loops of small intestine were matted together with plastic exudate, and upon separation of these loops collections of pus were found. A large collection of pus almost equal in size to that on the right was found in the left iliac fossa. A large cul-de-sac abscess was continuous with the abscess on the right. Another significant finding was a beginning empyema of the left pleural space. Anatomic diagnosis established at autopsy was ruptured gangrenous appendicitis, generalized peritonitis with multiple localized collections of pus in the peritoneal cavity, hydrothorax, and early empyema of the left pleural cavity and parenchymatous degeneration of the liver.

Comment.—This case represents that unfortunate circumstance in which, after partial localization of an appendiceal abscess, further peritoneal spread ensues. It did not seem desirable to operate upon this patient when he was first seen, although some localization was present. Drainage was undertaken in a desperate effort to overcome the evident spreading of the original appendiceal abscess. The almost unbelievable extent of suppuration in the peritoneal cavity as seen at autopsy leads one to wonder why the patient lived even a few hours.

In all of these 52 cases delay had ensued before consulting us. Many had been treated injudiciously with cathartics and other home remedies. In every case the infection had extended beyond the appendix, and each one presented an individual problem as to treatment.

As a rule the defensive forces operative in the peritoneal cavity are at least partially successful in walling off an acutely inflamed appendix before perforation occurs. The omentum migrates to surround the inflamed viscus and fibrinous adhesions quickly form. A definite degree of localization, therefore, usually exists at the time perforation occurs. If these cases are first seen after perforation and at the beginning of localization, we believe that great harm will be done by immediate operation. If given an opportunity, complete localization will follow and resolution will even occur in many cases. If, however, the patient is seen only a few hours after rupture of the appendix into virgin peritoneum, immediate operation is done, as has been stated above. If this same type of case is seen later after localization is beginning conservative treatment should be instituted. Conservative treatment of appendiceal peritonitis is indicated in those cases seen "too late for an early operation and too early for a late operation."[62]

By conservative treatment of appendiceal peritonitis, attempt is made to assist the physiologic response of the peritoneum in limiting the spread of the infection and favoring localization. Normally, cessation of intestinal peristalsis will occur, and treatment can best be directed toward promoting this physiologic reaction. The presence of vomiting and reverse peristalsis usually serves to empty the upper intestinal tract. By duodenal drainage with a perforated catheter,

this process can be greatly facilitated. This means of decompressing the upper intestinal tract assists also in relieving nausea or vomiting and in addition protects the intestine from the danger of excessive dilatation. Obviously catharsis will increase the spread of infection by stimulating peristalsis and will also retard localization by destroying limiting fibrinous adhesions. Because nothing can be given by mouth, fluids and electrolytes must be administered by some other route. Small amounts can be given by proctoclysis, but we rely almost entirely upon intravenous and subcutaneous administration. As a rule at least 3,000 c.c. of fluids are given daily. Almost routinely we use solutions of glucose and sodium chloride, given by intravenous injection.

In the administration of fluids we have been guided largely by the excellent work of Coller.[63-65] We owe much to the pioneer research of Coller and Maddock, whose study has placed the control of dehydration on a scientific basis. In 1936 they[66] undertook an intensive analysis of water metabolism in the sick and found that surgical patients lose about 2,000 c.c. of water by vascularization, and a minimum output of 1,500 c.c. of urine daily is necessary to insure adequate renal function. From these findings they deduced that patients who cannot take fluids by mouth require 3,500 c.c. of fluid daily. If the patient is losing fluid abnormally, as in vomiting, diarrhea, etc., provision should be made to replace this fluid loss also. They then studied a dehydrated patient and found that a patient in this condition weighing 60 kg. loses 3,500 c.c. of fluid. They estimated that a dehydrated patient should be given, in addition to the regular amount of fluid given a surgical patient, enough more to equal 6 per cent of the patient's body weight, or, for a 60 kg. patient, 3,600 c.c. plus 2,000 c.c. for vascularization and 1,500 c.c. for urine, or a total daily fluid administration of 7,100 c.c. In discussing the type of solution to be administered, Coller states that a saline solution should be given to replace fluid loss from the gastrointestinal tract and the remainder of the fluid should be 5 per cent dextrose in distilled water.

The patient should be placed in the Fowler position. We do this routinely to favor gravitation of the fluid exudate to the pelvis, for a residual abscess in the cul-de-sac of Douglas is much more readily diagnosed and treated than abscesses in the upper abdomen. Heat in some form should be applied to the abdomen. As a rule we use large hot fomentations together with a heat tent. If the weight of the hot fomentation causes any discomfort, the heat tent alone is used. There is evidence to show that heat aids in controlling distention by decreasing secretion in the intestine. Also abdominal pain seems to be relieved, at least somewhat, by the application of heat. Morphine should be given in fairly large doses. A. J. Ochsner originally used morphine because he believed it splinted the bowel. It has been shown

conclusively, however, both clinically and experimentally[67] that morphine does not retard peristalsis but is beneficial because it increases the tone of the bowel and prevents excessive dilatation of the intestine with its concomitant intramural compression of the vessels.

It is a great satisfaction to observe the change in the patient's condition soon after conservative treatment is instituted. Abdominal distention diminishes, nausea and vomiting subside, pain decreases, the pulse improves, and the fever becomes lower. As localization progresses, usually a palpable mass can be outlined. Optimum time for operation must be chosen. Operation too early will disseminate infection from an imperfectly localized abscess, and operation too long delayed may in a few cases allow intraperitoneal rupture of a localized abscess. Frequently localized peritonitis may subside spontaneously. With the formation of an abscess, however, evidence of a septic process continues. The leucocyte count decreases but still remains above normal. Fever continues but is usually very slight except for an afternoon elevation. Drainage of the abscess is indicated and carries a very good prognosis. Unnecessary manipulation in an attempt to remove the appendix should be avoided. If the abscess has been well localized and gentle handling practiced, it is generally possible to remove the appendix without disseminating the infection. With but 4 exceptions in this series of 52 cases, the appendix was removed at the primary operation. In 3 of these cases the patient returned in a few months for an interval appendectomy, and the other patient died. The type of incision employed depended for the most part upon the site of the appendiceal abscess. We have found that the pararectus incision with a retraction of the rectus muscle medially allows adequate exposure of the right iliac fossa with a minimum destruction of important structures of the abdominal wall. This type of incision was used most frequently, although the McBurney incision was used in 5 instances. In addition to the limitation of exposure afforded by the McBurney incision, we believe that drainage is hampered somewhat by this gridiron type of incision. Upon opening the peritoneal cavity, utmost care is maintained to prevent soiling of uninvolved peritoneum. This is best accomplished by adequate exposure and the careful placing of gauze packs. Before opening the abscess the wound edges should be protected likewise to avoid undue contamination of the abdominal wall. With one exception Penrose drains were used. The incision is closed very loosely with interrupted sutures, and in a few instances in which it was deemed advisable to close the abdominal incision, little or not at all, iodoform gauze was placed just down to the peritoneum to prevent a herniation of intestine through the incision. This gauze was not used as a drain in any way and was removed after twenty-four hours.

The performance of a primary cecostomy has been advocated by a number of surgeons for many years.[7, 68-70] In many of these difficult cases in which we found that, in spite of continuous duodenal decompression by the Wangensteen suction, the terminal ileum, cecum, and ascending colon were markedly distended, we have frequently inserted a catheter through the appendiceal stump after removal of the appendix. This cecostomy does not prolong the operation at all. We routinely remove the appendix by using the inversion without ligation technique.[71] This is the method that A. J. Ochsner introduced at the Augustana Hospital, and we have found no occasion to vary the technique. By this method the mesoappendix is first grasped with forceps, divided, and then ligated with fine catgut ligatures. Next, a purse-string suture of silk on a fine needle is introduced so as to include an intramural branch of the appendicular artery. This is accomplished by starting the purse-string suture from one side of the mesoappendix and passing it down to and including the submucosa, and bringing it out on the opposite side of the mesoappendix. The suture is then reinserted in exactly the same manner, thereby forming a loop around a segment of the cecal wall adjacent to the mesoappendix. This loop encircles any intramural branch of the appendicular artery and, after tightening the loop sufficiently to occlude the artery, the purse-string suture is continued around the cecum in the conventional manner. Next, three Ochsner clamps are applied to the base of the appendix, and, after walling off the area with moist sponges, the appendix is divided between the upper and middle forceps. We use a scalpel to section the appendix. There would be some advantage in using the thermocautery, but at the present time the type of anesthetic gas employed so frequently prevents the use of the cautery that we have continued to follow A. J. Ochsner's example of using only the scalpel. After the appendix is sectioned, a sponge is used carefully to wipe off the small amount of secretion on the middle forceps. The middle clamp is then removed and the crushed tip securely grasped with a pair of plain tissue forceps. Traction is then made upward on the fixed end of the purse-string suture and upward traction is also made on the cecal wall by grasping the tenial band with a toothed tissue forceps at a point opposite to the mesoappendix. The remaining Ochsner clamp is then removed and the stump of the appendix inverted into the lumen of the cecum by pushing it downward with the smooth tissue forceps. The purse-string suture is drawn tightly as the tissue forceps is gently freed and withdrawn. At this point, if a cecostomy is to be done, a 22 F catheter is passed through the lumen of the inverted stump and the purse-string suture tied securely. A second purse-string suture is then placed and the stump inverted further. This technique of cecostomy (Figs. 1-4), which was used in 35

cases, is very simple and provides a serosal tract which quickly closes upon removal of the cecal catheter. The tip of the catheter is directed either into the cecum or through the ileocecal valve into the terminal ileum, the choice being established by the indication for decompression. This catheter is brought out through the abdominal wall by

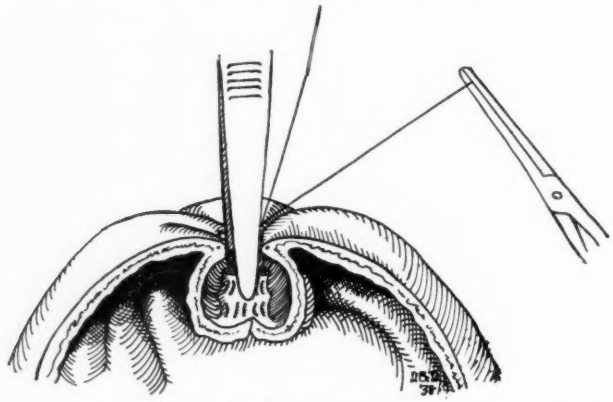

Fig. 1.—Appendiceal stump inverted without ligature, purse-string suture tightened slightly but not tied.

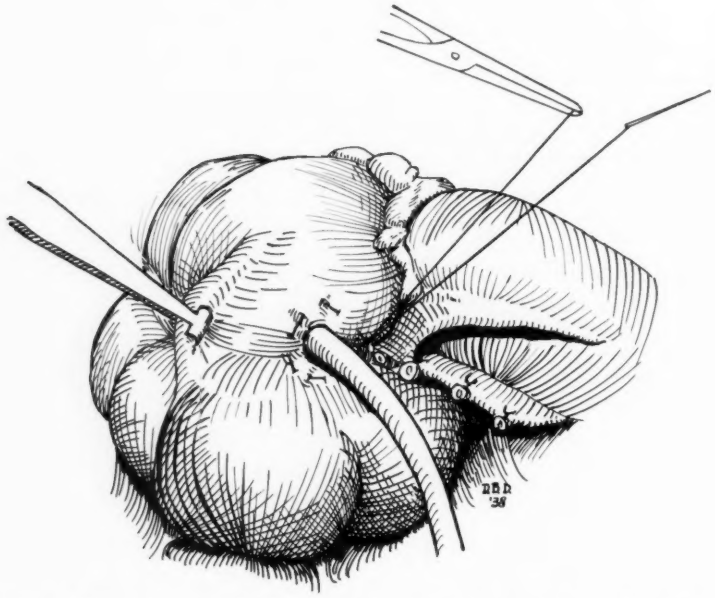

Fig. 2.—Catheter being pushed through lumen of inverted appendiceal stump.

means of a small stab wound which is so placed that the head of the cecum lies easily against the point of exit of the catheter. Through this catheter normal saline solution is injected at hourly intervals. By injecting saline, an opening in the catheter is assured, and, as a part of the solution will siphon away, drainage of fecal material and gas is facilitated. It is gratifying to note the absence of distention when

this type of cecostomy is used. The cecal catheter is removed usually after ten days (the average interval was 10.6 days in our series). Its use, however, does not replace duodenal suction, as this is employed concomitantly.

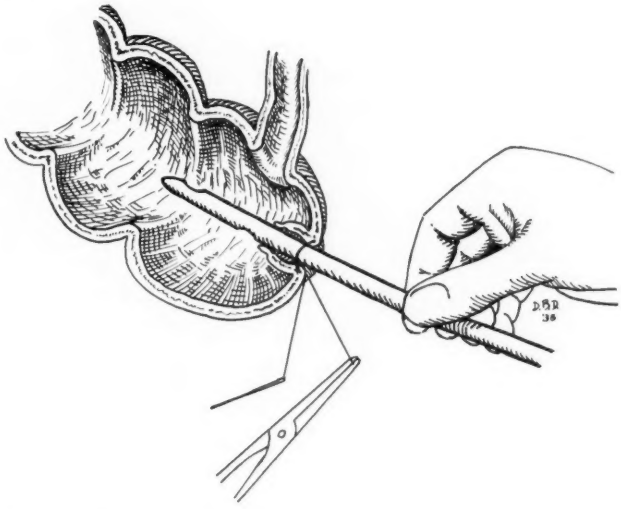

Fig. 3.—Catheter in cecum, first purse-string suture being tied.

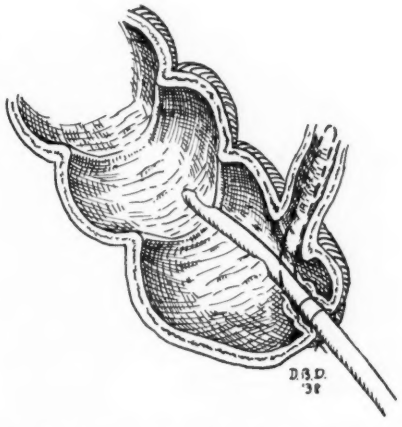

Fig. 4.—Second purse-string suture tied. Dotted outline shows alternate position of catheter in ileum.

According to Paine and Wangensteen,[72] Ward, of San Francisco, was apparently the first to treat postoperative distention by employing the principle of continuous suction in connection with the in-lying duodenal catheter. They[72] state, however, that Westermann[75] and Kappis[76] more than twenty-five years ago first advocated the use of the duodenal tube in the treatment of distention of peritonitis. The general technique of employment which has been described by Wangensteen and Paine[73, 74] is fairly simple: A small duodenal catheter of

the Levine type is passed through the nose into the stomach. A simple water siphon exerts constant suction (about 75 c.c. of water). Perforations are carried back on the catheter for about ten inches, which provide for simultaneous suction of both the stomach and duodenum when the tip of the catheter enters the duodenum. This prevents further vomiting.

Postoperative treatment is essentially a continuation of the conservative treatment used preoperatively. The patient is placed in the Fowler position or is turned to the right side if this best facilitates drainage. Adequate doses of morphine are given, and attention to water balance is observed, chiefly by the use of intravenous solutions of glucose and sodium chloride. As a rule heat in the form of a heat tent is kept constantly applied to the abdomen. The average duration of the postoperative hospital stay was 25.3 days. There were 3 deaths in this series of 52 cases of perforated acute appendicitis. An analysis of 1 has been given above and discussion of the other 2 cases follows:

CASE 3.—W. J., white male, 40 years of age, truck driver. Two days prior to admission the patient developed a "stomach-ache" characterized by a dull, dragging pain across the abdomen. The ingestion of some pills resulted in nausea and vomiting. The following day the pain localized in the right lower quadrant. The patient no longer vomited but suffered abdominal distention. Upon admission Jan. 25, 1932, the patient was placed on routine conservative treatment for three days. At this time there seemed to be sufficient evidence of localization to warrant operation. Accordingly, on the third hospital day an operation was performed. The appendix was gangrenous, filled with pus, and surrounded by omental and peritoneal adhesions. There was considerable free pus and free fluid in the abdomen. Appendectomy with primary cecostomy was done and two cigarette drains were inserted. Following the operation, the patient's temperature rapidly rose to 108°, his pulse became weaker, and he expired on the afternoon of the first postoperative day. No autopsy was permitted.

Comment.—This case came to operation too soon and should have been treated conservatively for a longer period of time before operation. When at operation it was seen that there was inadequate localization of the inflammatory process, nothing should have been done except the simple insertion of drains. Considerable free fluid containing plastic exudate was seen in the peritoneal cavity, and this already existing diffuse peritonitis was made more virulent by dissemination of the infection from the appendiceal abscess.

CASE 4.—C. Q., an obese male, 61 years of age, electrician, admitted to the hospital June 12, 1935. When the patient became ill one week previously, he thought he had a "cold in his stomach." During the illness he complained of nausea and anorexia, and consequently ate very little. For constipation he took a "physic" pill daily. Abdominal examination upon admission revealed a mass the size of an orange in the right lower quadrant. After conservative treatment for two days, operation was performed. A large appendiceal abscess was found well walled off in the right lower quadrant. The appendix, which was large, swollen,

and gangrenous, was removed and a catheter was inserted into the cecum through the appendiceal stump. Six cigarette drains were inserted into the appendiceal abscess. Routine postoperative treatment was instituted. The patient's immediate postoperative condition was good. On the first postoperative day the patient was doing rather well and there was no distention. On the second postoperative day the temperature became normal and the patient's condition appeared excellent. During the night of the third postoperative day, the patient had a chill, developed chest pains, became dyspneic, and had marked tachypnea and a nearly imperceptible pulse. The temperature rose at once to 102°. Coarse râles could be heard over the entire chest. The patient was placed in an oxygen tent and digitalis was given intravenously. Cyanosis became noticeable in spite of oxygen. By the fourth postoperative day the temperature had reached 105.6°. The pulse rate varied from 140 to 160. The heart was irregular, consolidation was evident in the bases of both lungs, and respiration was labored. Pulmonary edema increased and on the sixth postoperative day the patient died.

Comment.—This obese patient was an elderly alcoholic who developed a diffuse bronchopneumonia with rapidly increasing pulmonary edema and cardiac failure. Unfortunately autopsy was not allowed, but clinically it seemed that the abdominal condition was definitely improving at the time of onset of the pneumonia.

During the corresponding period of time represented by our series, there were treated at the Augustana Hospital 3,771 cases of appendicitis (Table III). Of this total number, there were 83 deaths, a mortality of 2.2 per cent. It is interesting to note the coincidence that the mortality percentage exactly corresponds to the percentage quoted by A. J. Ochsner in 1904 from the Augustana Hospital. The highest percentage of these deaths occurred, as would be expected, in acute appendicitis and in particular in those cases with perforation. There were 23 (19 per cent) deaths in this group. In general, the Augustana Hospital series parallels our series. It would be difficult to express an opinion concerning the extent of preoperative preparation in all of those which required delayed treatment. It would undoubtedly be found that a few were operated upon prematurely and incompletely prepared. In general, however, most of these patients were treated conservatively and with the exception of some modifications received routine Ochsner treatment. It is favorable to note the lowest percentages were met in 1937 and 1938. More adequate supervision and more careful preoperative preparation may accomplish a still further lowering of the mortality.

Of the 195 cases of acute appendicitis in our series, 143 cases had immediate operation and 52 patients were treated by the deferred operation. In the series treated by immediate operation, there was 1 death, a mortality of 0.69 per cent. In those cases that were operated upon at once, the infection in nearly every case was limited to the appendix. In a few cases extension had occurred so recently that no adhesions had formed. In the series treated by deferred opera-

TABLE III

MORTALITY ACCORDING TO YEARS IN AUGUSTANA HOSPITAL SERIES

APPENDICITIS	1931		1932		1933		1934		1935	
	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS
Chronic	398	7	325	10	334	4	327	5	269	0
Acute, simple	220	0	193	1	173	5	111	1	125	3
Acute, perforated with abscess	10	0	13	2	10	2	18	1	10	1
Acute, perforated with peritonitis	28	2	23	4	11	2	18	7	21	5
Total	656	9	554	17	528	13	474	14	428	9
Mortality (per cent) by years	1.37%		3.07%		2.46%		2.91%		2.10%	

	1936		1937		1938		TOTAL		MORTALITY %
	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	
Chronic	263	7	288	2	173	1	2407	36	1.49
Acute, simple	139	1	117	0	59	0	1140	11	0.96
Acute, perforated with abscess	11	1	20	4	11	2	103	13	12.61
Acute, perforated with peritonitis	20	3					121	23	19.00
Total	433	12	425	6	243	3	3771	83	2.2
Mortality (per cent) by years	2.77%		1.40%		1.23%				

tion, there were 3 deaths, a mortality of 5.7 per cent. These were all seen "too late for early operation." In all but 1 case in this series drainage was used. In 4 cases the appendix was not removed at the primary operation. In the series of 195 cases of acute appendicitis there was a mortality of 2.05 per cent. Contrary to the usual sex distribution there were more females than males in our series (106 and 89 respectively). The 4 deaths in our series occurred in men, a mortality of 4.5 per cent. The oldest patient with acute appendicitis was 72 years old and the youngest was 5 years of age. The greatest number of patients was in the second, third, and fourth decades, there being 59, 50, and 31 patients in these respective groups. There were 19 patients in the fifth decade, 3 of whom (aged 41, 46, and 48 years) died, a mortality of 15.9 per cent. In all, only 4 patients in the seventh decade were seen, and 1 of these died (a mortality of 25 per cent). The average leucocytosis met with in our series of acute appendicitis was 17,500 (15,200 in simple acute appendicitis and 19,900 in ruptured appendicitis). The red blood cell count was not remarkable, as would be expected, the average being 4,571,000. In the cases with simple acute appendicitis the average maximum temperature was 99.8°, with 6.3 days being the average febrile period; whereas, in the cases of acute ruptured appendicitis the average maximum temperature was 102.5° with an average febrile period of 19.2 days. This difference in febrile reaction in these two groups emphasizes somewhat the definite variation in the severity of the disease. In the more severe manifestation of the disease, a need for adequate exposure made us favor the use of a muscle retracting right rectus incision. In those cases treated by delayed operation, a McBurney incision was used only 5 times. One of these patients died as detailed above. A right rectus incision was made in the 3 other patients who died after being treated by delayed operation. When immediate operation is indicated, we have a tendency to favor the McBurney incision unless we anticipate a need for more than the usual exposure. Consequently, we have used the McBurney incision in 78 of the 143 cases of this series. In this immediate operation series the 1 patient who died had a right rectus incision.

COMMENT

We have reported this series of cases observed during the past seven-year period in order to review our results from the conservative treatment of acute appendiceal peritonitis. These results seem encouraging enough to justify the continuation of this type of management. From a review of the 4 deaths occurring in this series, it is probable that 1 of them resulted from inadequate preoperative preparation. This was undoubtedly due to misjudging the degree of localization present. It is reasonable to believe that this case could have

been saved by awaiting further localization before operation or by limiting the procedure to simple drainage. One death (H. A., Case 2) occurred when a partially localized appendiceal abscess spread further, to cause a very extensive suppurative diffuse peritonitis. It is difficult to see how any type of treatment could have altered the outcome in this case. The other 2 deaths were caused by factors other than those directly due to appendicitis and might be expected to occur in any group of surgical cases.

In our series, as in nearly all other reports, the cases presenting the most difficulty in treatment were neglected cases that had received cathartics. As of course would be expected, many complications were encountered. Pylephlebitis occurred in 1 case and was the most serious complication met with in the patients who recovered. In 2 cases there was a rupture of a localized appendiceal abscess. One of these patients died. These complications will be completely discussed in a paper now in preparation.

Departure from routine Ochsner treatment consisted only in minor refinements in therapy. The introduction of duodenal drainage popularized by Wangensteen has been of great benefit in the treatment of abdominal distention so frequently encountered in complicated appendicitis. Cecostomy done at the time of operation has seemed to have been of further help in a few of the most desperate cases by aiding in the decompression of the lower small intestine, cecum, and ascending colon. Maintaining the fluid intake of these patients is more readily accomplished at this time than when Ochsner originally advocated this plan of treatment. We believe that far from being discarded the Ochsner treatment should receive further consideration and evaluation. If such is done, we feel certain that a more universal adoption of this plan of management will be made.

SUMMARY

1. A series of 303 cases of appendicitis are herein presented.

2. In 195 cases of acute appendicitis the principles of the Ochsner treatment were employed.

3. A plea is made for a review of the conservative treatment of appendiceal peritonitis.

4. In our series of 303 cases there were 4 deaths, a mortality of 1.3 per cent.

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ACUTE APPENDICITIS, SIMPLE AND COMPLICATED

A Critical Analysis of 1,010 Cases Treated at the Robert Packer Hospital

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IN AN editorial in *The American Journal of Surgery* for August, 1935, Guthrie expressed the opinion of thoughtful surgeons that most reported grouped statistics on appendicitis meant little because they were all-inclusive and varied depending principally upon the number of chronic, acute clean, or even secondary cases included in the series. It is evident that on a careful surgical service there will be only an occasional accidental death in those cases where the pathology is limited to the appendix per se. However, in the remaining cases in which the pathology has spread through perforation to the stage of local peritonitis, spreading or diffuse peritonitis, or abscess formation, the mortality will be necessarily high. It is by a careful study of these cases that the merits of various treatments and the skill of surgeons may be evaluated.

There have been several recent reports in which an honest attempt has been made to state a mortality in these complicated cases. The figures have been high. It is in the spirit of self-analysis that we are reporting a study of 1,010 cases of acute appendicitis handled on the surgical service of the Robert Packer Hospital during the five-year period from June 1, 1932, to May 31, 1937. Two hundred and sixty-six cases with the diagnosis of subacute appendicitis and 279 chronic cases operated upon during this period are not included. In an attempt to aid diagnosis, certain statistical compilations regarding symptomatology and findings in this group of cases are also reported.

Connell has recently suggested that for statistical study acute appendicitis cases be analyzed according to the following classification:

Acute { Nonperforated
Perforated { Abscessed
Peritonitis { Local
Diffuse

Following this classification these 1,010 cases may be subdivided as shown in Table I. It is evident that less than 20 per cent of the cases in the series make up the group of perforated cases in which, as will be shown, most of the mortality falls.

Predisposing Factors.—Before proceeding to a discussion of the mortality in this series, several predisposing factors of appendicitis and its

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TABLE I

GENERAL GROUPING

			NUMBER OF CASES	PERCENTAGE OF SERIES
Nonperforated (81.28%)	821	Acute	670	66.33
		Gangrenous	151	14.95
Perforated (18.72%)	189	Abscessed	68	6.78
		Local peritonitis	111	10.95
		Diffuse peritonitis	10	0.99
		Total	1010	100.00

complications and some information concerning the symptomatology of these cases are to be considered.

Age.—Although appendicitis may occur at any age, it is known to have its highest incidence in the "teens." As stated by Deaver: "The susceptibility of young persons to appendicitis is mainly due to the abundance of lymphoid tissue and the tendency to infection of all lymphoid tissue during adolescence and to the dietary indiscretions that are the privilege of youth." The average age for the series, calculated on a basis of 960 cases in which age was given, is 23.8 years. The average for simple acute cases is 21.6 years; for gangrenous, 27.7 years; for abscessed, 28.13 years; and for the local and general peritonitis group, 29.6 years. With an increase in the severity of the disease, there is an increase in the average age. Of more interest is the increased proportion of patients in the extremes of life; i.e., 6 years or under and 50 years or older in the complicated groups as compared to the simple acute cases as shown in Table II.

TABLE II

INCIDENCE IN EXTREMES OF LIFE

	TOTAL NO. CASES	NUMBER 6 YR. AND UNDER	PERCENTAGE 6 YR. AND UNDER	NUMBER 50 YR. AND OVER	PERCENTAGE 50 YR. AND OVER
Acute	642	11	1.71	29	4.52
Gangrenous	134	6	4.48	19	14.18
Abscessed	68	6	8.80	13	19.11
Perforated with local or diffuse peritonitis	116	4	3.45	24	20.70

This tendency is shown even more clearly by graphs based on the five-year age groupings. The curve in simple acute cases reaches a high at 16 to 20 years and is very low in 0 to 5 and 51 plus groupings. In the complicated cases, including gangrenous appendicitis, the height of the curve is, of course, between 11 and 20 years, but the curve starts at a higher level and is maintained at a higher level in the older age groupings.

Sex.—Most reported series give a higher incidence of appendicitis in the male than in the female. This is true in our series which includes 529 males and 481 females. Deaver has emphasized the importance of

the better blood supply in the female afforded by the appendiculo-ovarian artery. This anatomic difference may be of even more significance in determining what happens to the patient who develops appendicitis.

TABLE III

SEX GROUPING

	MALE	FEMALE
Acute	311	359
Gangrenous	102	49
Abscessed	43	25
Perforated	73	48
Total	529	481

It will be noted from Table III that the cases progressing to gangrene and perforation were predominantly male. Ten patients in the series had diffuse peritonitis, 9 of these were males. Of the 21 deaths in the series, 13 were males. It is likely that this greater tendency for males to develop complications is due not alone to anatomic differences, but also to a greater tendency to procrastination in seeking medical aid and continued physical exertion in the presence of developing appendicitis.

Seasonal Incidence.—It has seemed in the work of this clinic that there is some difference in the number of cases of appendicitis seen in the different seasons. The monthly incidence of cases is given under the diagnosis in Table IV.

It is shown that there is a definite increase in cases in the spring and summer. The total number of 531 cases from March 1 to Sept. 1 as compared with the 465 cases from Sept. 1 to March 1 shows an increased incidence during the warm months of about 15 per cent over the fall and winter.

TABLE IV

SEASONAL INCIDENCE

MONTH	ACUTE	GANGRENOUS	ABSCESSED	PERFORATED	TOTAL
January	65	6	5	15	91
February	57	10	9	11	87
March	57	18	4	10	89
April	64	13	10	7	94
May	60	12	3	7	82
June	75	16	2	11	104
July	42	14	9	15	80
August	56	14	10	16	96
September	48	12	4	7	71
October	58	15	4	9	86
November	53	9	2	6	70
December	35	12	6	7	60
Total	670	151	68	121	1,010

This coincides with the greater prevalence of gastrointestinal disorders during these seasons, and in the absence of a better explanation this will be assumed to be the cause.

TABLE V

SEASONAL GROUPING

INCLUSIVE MONTHS	NUMBER OF CASES	
March-May	265	545
June-August	280	
September-November	227	465
December-February	238	

Laxatives.—In recent years the profession, and in more recent years the public, have been made cognizant of the role of laxatives in increasing the complications of appendicitis. Despite this fact, a careful history brings out the information that a large percentage of even the most recent cases in the series had laxatives. In this series 560 case histories gave definite information as to cathartic administration; 293, or over 50 per cent, had had one or more doses of laxative. The pioneer work of Bowes in publicity is certainly not in vain, but his message should be broadcast over the land. Even more tragic is the notation occasionally seen, "his physician gave him a laxative." Table VI brings out the fact that, whereas only 43 per cent of the simple acute cases had cathartics (in specified records), 60 per cent of the gangrenous had laxatives, and in cases in which the pathology had extended beyond the confines of this organ over 74 per cent had had laxatives. In other words, comparing the number of cases with laxative intake to those without in the simple cases, the ratio is 3:4; whereas in the perforated cases it is 3:1; or the proportion of perforated cases having laxatives is in the ratio of 4:1 as compared with the simple acute cases. This only further confirms the opinions already expressed that cathartics remove Nature's splint on the intestinal tract and increase dramatically the number of cases which go on to perforation.

TABLE VI

LAXATIVE INCIDENCE

TYPE OF CASE	LAXATIVE	NO LAXATIVE	NOT SPECIFIED	PER CENT SPECIFIED WITH LAXATIVE
Acute	148	200	322	43
Gangrenous	54	36	61	60
Abscessed	37	10	21	78
Local peritonitis	48	19	44	72
Diffuse peritonitis	6	2	2	75
Total	293	267	450	52

Time From Onset to Perforation.—Undoubtedly another most important factor predisposing to complications of appendicitis is the increased time between the onset of symptoms and the operation. This is borne out in this study. Table VII demonstrates that the complicated cases were the ones with a longer average time between onset and operation.

The average time for the cases with confined pathology, i.e., acute and gangrenous, was about 36 hours. The time for perforated cases was figured with the exclusion of those cases in which delayed operation or no operation was performed, so the difference between 51 and 36, or 15 hours, is delay time pure and simple.

TABLE VII

TIME UNTIL OPERATION

	NO. OF CASES WITH TIME GIVEN	AVERAGE TIME	LONGEST TIME	SHORTEST TIME
Simple acute	648	36.92 hr.	250 hr.	3 hr.
Gangrenous	144	33.00 hr.	240 hr.	3 hr.
Abscessed	66	13.1 days	35 days	30 hr.
Perforated with local or diffuse peritonitis	98	51.0 hr.	144 hr.	5 hr.

In this series this delay of 15 hours is the critical time during which, in the average case, perforation took place. The figures in the abscessed cases do not represent delay time in their entirety, since most of these patients were treated conservatively for a time to further localize the abscess. However, the time between onset and admission to the hospital was necessarily long, because at the time of admission it was considered that Nature had already, in part at least, walled off the infectious process.

SYMPTOMATOLOGY

It might be of some value in a review of a series of this size to analyze the common symptoms of appendicitis and its complications. Deaver states that: "Pain, tenderness and rigidity are the three cardinal symptoms of appendicitis; they are usually associated with vomiting, fever and leucocytosis." In this study the variables which could be most accurately evaluated from the charts have been considered. This includes the position of onset pain, admission temperature, leucocytosis, and differential. No attempt to analyze the degree of pain has been made because it is subjective and a statement of its quantity is not wholly dependable. No analysis of tenderness and rigidity has been made, not because they are not important, for they are most important, but because their evaluation introduces a personal equation and is likely to be inaccurate unless the same surgeon examined each patient, which was not the case.

The point of onset of pain is given by most authors as epigastric or periumbilical. This may be classical but by no means invariable. The incidence of the various points of onset is given in tabulated form (Table VIII) as noted by the physician taking the history. As would be expected, there is no significant difference between the cases as regard point of onset pain and ultimate pathology, since the cases all start as acute appendicitis.

TABLE VIII

ONSET PAIN

POSITION OF ONSET	ACUTE	GANGRENOUS	ABSCESSED	PERFORATED	TOTAL
Epigastric	165	39	25	43	272
Generalized	104	35	15	25	179
Right side	30	2	2	3	37
Left side	0	0	0	1	1
Right lower quadrant	244	34	8	19	305
Right upper quadrant	5	1	2	0	8
Left lower quadrant	2	2	0	3	7
Umbilical	56	18	4	8	86
Upper abdominal	9	5	3	1	18
Lower abdominal	39	15	8	14	76
Not given	16	0	1	4	21
Total	670	151	68	121	1,010

The largest number of patients, about 30 per cent, gave the point of onset pain as the right lower quadrant. About 27 per cent gave the classical epigastric pain and 18 per cent stated the original pain was generalized, with only about 8.5 per cent periumbilical.

The sequence of pain-vomiting is pathognomonic of acute appendicitis, but vomiting is by no means constant. In the simple acute cases only 47 per cent had nausea and vomiting. An additional 33 per cent had nausea without vomiting. It is significant, however, that with increasing severity of the pathology present there was an increasing number of patients who had vomiting as a symptom, increasing to 72 per cent in cases of local peritonitis and 80 per cent in cases of diffuse peritonitis; 54.1 per cent of the entire series vomited.

Fever is a variable sign. It may be stated that a high fever usually indicates a serious condition, but the converse is by no means true. Average temperatures would seem, from this study, significant and roughly an index to the pathology, since the average in simple acute cases was 99°; whereas, with gangrene the average was 99.44° and rose in abscess and peritonitis cases to above 100.5°; the large range between the highest and lowest temperatures given for various types, a range of almost 8° for the series, shows the variability of this sign.

Leucocytosis is also variable and a treacherous sign at times because of the unwarranted reliance placed in it by some men. Here again the averages in this series are typical, showing an increasingly high count with increasing pathology, the average ranging from 12,057 leucocytes in the simple acute cases to 17,728 in the cases of diffuse peritonitis. Here again, however, the range is so great in cases with comparable pathology that one is forced to conclude that it is of value only as one of a number of signs and symptoms and it must be disregarded when at variance with the rest of the picture. Attention must be called to the fact that often a low count in the presence of grave clinical symptoms is a danger signal and regarding it as anything else is likely to lead to disastrous results.

Many consider the differential count of especial significance as an indication of the severity of the infectious process. Without doubt it may be said that it is less often misleading than the total count, but when occasional patients are seen with definite gangrene of the appendix and a polymorphonuclear percentage of 48 or a perforated case with 55 per cent polymorphonuclears it is evident that the differential is not "fool proof" either. On the average, however, the percentage of polymorphonuclears will increase dependent on the severity of the process; this was borne out in our average figures.

In the experience of this clinic by far the most dependable symptom as to degree of infection is the severity of pain, and the most reliable sign is tenderness over the appendix. Usually tenderness will be found in the right lower quadrant in the vicinity of McBurney's point, but occasionally, in a low-lying or retrocecal appendix, tenderness is best demonstrated on rectal examination, because the palpating finger here comes in closest proximity to the involved organ.

MORTALITY

It is difficult to comprehend that in spite of new diagnostic aids, in spite of better trained surgeons, in spite of more meticulous technique and increased accessibility of hospitals, the mortality in appendicitis has been increasing for thirty years; but such is the case. Vital statistics, carefully compiled by the census bureau and life insurance companies, have shown a small but progressive rise in the mortality from appendicitis year by year. This is the fact that makes any critical analysis of cases worth while; this is the fact that stands as a challenge to each present-day surgeon; this is, as Royster has so aptly dubbed it, "the tragedy of appendicitis."

An honest effort has been made in this study to determine the pathologic condition present and responsible for the deaths which occurred. This mortality study is not made to flatter the "ego" of this clinic group, although the figures are comparatively good, but to point out the types of cases in which the deaths are occurring as a stimulus to more thoughtful care of these patients.

In the five-year period analyzed, 1,010 cases were diagnosed as acute appendicitis, simple or complicated. With few exceptions this diagnosis was checked by operation or necropsy. In this series there were 21 deaths, a gross mortality of 2.08 per cent. That this figure means little has been stressed, therefore it will be "broken down." There were 670 simple acute cases and 151 gangrenous cases. This gives a total of 821 cases in which the process was limited to the appendix per se; in this group there were 3 deaths, or a mortality of 0.365 per cent. The remaining 189 cases include the perforated ones with local peritonitis, general peritonitis, or a walled-off appendiceal abscess. Where the pathology had extended beyond the confines of the appen-

dix, there were 18 deaths, a mortality of 9.52 per cent. A further analysis of mortality as to pathologic process present is presented in Table IX.

TABLE IX

MORTALITY IN RELATION TO PATHOLOGY PRESENT

TYPE OF CASE	TOTAL NUMBER	NUMBER OF DEATHS	MORTALITY IN PER CENT	
Acute	670	1	.15	
Gangrenous	151	2	1.32	
Abscessed	68	5	7.14	9.52%
Perforated with local peritonitis	111	7	6.30	
Perforated with diffuse peritonitis	10	6	60.00	
Series	1,010	21	2.08	

The one death in the simple acute cases was in an obese girl, 17 years of age, with Fröhlich's syndrome who developed bronchopneumonia and peritonitis postoperatively. Both gangrenous cases developed postoperative intestinal obstruction, one having also a pelvic abscess. Of the patients with appendiceal abscesses, 3 died following drainage of the abscess, 1 died of peritonitis following the secondary appendectomy, and 1 died without operation. Of the 7 deaths in the patients with local peritonitis, 4 progressed to diffuse peritonitis postoperatively and 1 died without operation. The 6 deaths in the diffuse peritonitis group are self-explanatory.

It is of interest to compare these figures with other statistics reported in the literature. It is impossible to tabulate all of this information in comparative form because of the varying classifications used, therefore several reported series will be discussed briefly.

Bancroft recently reported a group of 350 cases from the Fifth Avenue Hospital, New York City. There were 15 deaths. There were no deaths in 129 cases showing no adjacent peritoneal reaction; 5 deaths, or 3.3 per cent mortality, in 148 cases with free fluid; a mortality of 4.3 per cent in 46 cases of abscess; and 100 per cent mortality in 8 cases of diffuse peritonitis.

Christopher and Jennings reported, in 1932, a group of 1,138 cases from Evanston Hospital with 47 deaths. Mortality in 955 nonperforated cases was 1.78 per cent; in 183 perforated cases, it was 16.39 per cent.

In 1934 Keyes reported a series from Barnes Hospital, St. Louis. The mortality in 246 simple acute cases was 2.44 per cent; in 193 abscessed cases, 6.22 per cent; and in 56 ruptured cases with peritonitis, 19.64 per cent.

Schullinger reported an eighteen-year group, 1916-1933, at Presbyterian Hospital, New York City. His mortality in simple acute cases was 0.59 per cent in 1,175 cases. In 630 cases with no gross perfora-

tion, but fibrin on the appendix and some peritoneal exudate, probably comparable to our gangrenous group, the mortality was 1.9 per cent. Mortality in 571 abscesses was 10.5 per cent. In 329 cases of diffusing peritonitis the death rate was 17.02 per cent, while in 25 cases of generalized peritonitis it was 88 per cent.

Recently, King of Binghamton, N. Y., reported 364 acute cases comprising those operated in the years 1929 to 1930 and 1934 to 1935. In 248 uncomplicated cases there was but 1 death, or a mortality of 0.4 per cent. Cases of acute local peritonitis carried a mortality of 7.5 per cent; the 32 appendiceal abscesses, 12.5 per cent; and for 17 cases of diffuse peritonitis, 52.9 per cent. The group mortality in the complicated series was 15.5 per cent.

Among the series reported very recently is one by Totten, from the University of Southern California; one by McClure and Altemeier, from Ford Hospital, Detroit; and one by Kirtley and Daniel, of Vanderbilt University, Nashville, Tenn. The California series had a mortality of 4.2 per cent for 7,651 acute cases. The mortality in 6,607 nonperforated cases was 0.78 per cent and for a group of 1,251 perforated cases from July, 1921, to July, 1935, was 23.5 per cent. Ford Hospital reported a total percentage of 10.4 deaths in 252 perforated cases. This included 55 cases of local peritonitis with no deaths; 115 appendiceal abscesses, of which 4.2 per cent died; and 65 cases of general peritonitis and 15 cases of general peritonitis plus abscess, with mortalities of 21.5 per cent and 46.6 per cent respectively. Kirtley and Daniel reported 308 operated cases of perforated appendicitis with a mortality of 16.3 per cent; 4.2 per cent in 118 cases of appendiceal abscess, 8.1 per cent in 87 cases of local peritonitis, and 36.9 per cent in 103 cases of spreading or generalized peritonitis.

It is evident from only a glance at these figures that they are not entirely comparable. It is clear that since the percentages of diffuse peritonitis cases and local peritonitis cases vary so in respect to the total of perforated appendices varying criteria are being used for classification. There are only two comparable figures common to most of these series and these are the total mortalities for the acute cases and the mortalities for the perforated appendices including abscesses, local and general peritonitis. These figures are presented in Table X.

TREATMENT

It has been the practice in this clinic to operate upon all cases diagnosed as acute appendicitis nonperforated or upon those in which perforation is thought to have occurred recently, as emergencies. This is without doubt in many cases an unnecessary precaution, but certainly, in a large group of cases, it saves many from passing into the complicated group fraught with a much higher mortality. For a

TABLE X

COMPARATIVE MORTALITY

SERIES OF:	TOTAL ACUTE CASES	MORTALITY PER CENT	TOTAL PERFORATED CASES	MORTALITY PER CENT
Bancroft	350	4.2		
Schullinger	2,730	5.7	925	15.2
King	364	5.2	116	15.5
Christopher-Jennings	1,138	4.13	183	16.39
Totten	7,651	4.2	1,251	23.5
McClure and Altemeier			252	10.4
Kirtley and Daniel	994	5.14	308	16.3
Authors' series	1,010	2.08	189	9.52

number of years, Dr. Donald Guthrie, chief of our clinic, has been a staunch advocate of the Ochsner conservative treatment of those patients who have had perforations several days before being seen and who show a high fever, fast pulse, and abdominal distention. It is in these cases of developing or diffusing peritonitis that we feel the patient is given his best chance by means of Wangensteen drainage, Fowler's position, heat or ice to the abdomen, adequate intravenous chlorides, and physiologic rest of the intestinal tract by frequent administration of morphia. Beekman has pointed out the fact that in infants radical treatment is indicated despite diffusing peritonitis because of the inability of the small child's omentum and abdominal viscera to wall-off infection. In cases which have begun to localize an abscess when first seen, there can be little doubt of the treatment to be followed. These patients are treated conservatively until the temperature and pulse have receded and localization is complete. At this point they are drained and the appendix removed only if accessible. If the organ is not removed, the patient is strongly advised to return for secondary operation in six weeks to two months.

It is of interest in this connection that, of the 67 cases of appendiceal abscess operated upon in this series, it was deemed wise to remove the appendix in only 15, or 22 per cent, of the cases. Of the remaining 52 patients, 38 subsequently returned for secondary appendectomy. Of the 15 patients from whom the appendix was not removed primarily or secondarily in this clinic, it seems likely that a good proportion have subsequently been induced or forced by further infection to have it removed at some other institution.

The surgeons of the Guthrie Clinic have been slow to abandon the operation of enterostomy in complications of appendicitis because of the belief founded on experience that it still saves lives in certain cases doomed in spite of the yeoman's service rendered in many cases by Wangensteen decompression. That it is not necessary to resort to it so often as previously is not only admitted but attested to. In this series enterostomy was resorted to in 10 cases, or practically 1 per

cent of the series. This was complementary at the time of original operation in 6 cases and secondary, three to six days postoperative, in 4 cases. That the operation was of value is evident when we consider that 50 per cent of these distended, critically ill patients recovered.

TABLE XI

ENTEROSTOMIES

TYPE CASE	COMPLEMENTARY	SECONDARY	NUMBER DIED	PER CENT DIED
Gangrenous	0	2	2	100
Abscessed	3	0	2	66
Perforated	3	2	1	20
Total	6	4	5	50

Drainage.—In recent years the old dictum "when in doubt, drain" has become "when in doubt, don't drain." In accord with this trend we have found in the more recent cases of this series a decreasing number of drained cases, especially in the gangrenous cases and early perforations.

TABLE XII

DRAINAGE

TYPE OF CASE	TOTAL NUMBER	NUMBER DRAINED	PER CENT DRAINED	
Acute	670	9	1.3	
Gangrenous	151	37	24.7	
Abscessed	68	69	100.0	(2 operated upon twice; 1 not operated upon)
Perforated with local peritonitis	111	81	73.0	
Perforated with general peritonitis	10	3	30.0	
Total	1,010	100	19.7	

It will be seen by Table XII that less than 20 per cent of all the cases were drained; 73 per cent of the perforated cases were drained and about 25 per cent of the gangrenous cases.

Associated Conditions and Complications.—A number of associated conditions were present in cases of this series. Many were incidental conditions which bore no relationship to the primary condition and influenced it little. A few will be mentioned which may be of significance. Acute tonsillar infection was present in 5 cases. Some have felt coexisting tonsillar infection to be significant in etiology; its infrequency in this series proves nothing. Six patients in the series were diabetics. No deaths occurred in these cases, proving the safety with which acute surgery may be done in diabetics when carefully managed. Pregnancy was present in 3 acute cases which were operated upon without mortality to fetus or mother. Three of the patients were

tuberculous and 10 of the female patients had coexisting salpingitis. Two had Meckel's diverticula, asymptomatic and discovered accidentally.

The more important and frequently occurring complications are summarized in Table XIII.

TABLE XIII

COMPLICATIONS IN RELATION TO PATHOLOGY PRESENT

COMPLICATION	ACUTE	GANGRENOUS	ABSCESSED	LOCAL PERITONITIS	DIFFUSE PERITONITIS	TOTAL
Postoperative peritonitis	1	1	3	5		10
Postoperative intestinal obstruction	0	3	0	2	0	5
Pelvic abscess	2	0	4	3	1	10
Pneumonia	1	1	0	1	1	4
Atelectasis	1	0	0	0	0	1
Pulmonary embolus	0	2	0	2	0	4
Wound abscess	3	5	1	2	0	11
Hemorrhage	2	0	1	0	0	3

Spreading peritonitis postoperatively accounted for most of the postoperative deaths in the local peritonitis groups and in the appendiceal abscesses as well as the one simple acute case which ended fatally and one of the gangrenous. The incidence of other complications is also of interest. Intestinal obstruction occurred in 5 patients and 10 had pelvic abscesses. Pulmonary complications were infrequent and only 4 cases of embolus with 1 death occurred. Eleven wound abscesses occurred.

SUMMARY

A series is presented of 1,010 consecutive cases of acute appendicitis seen on the surgical service of the Guthrie Clinic and Robert Packer Hospital during the five years from June 1, 1932, to May 31, 1937. During this period there were 21 deaths, including nonoperated as well as operated cases. This gave a group mortality of 2.08 per cent for acute appendicitis in this clinic. Eighteen of these deaths were in the 189 cases in which the appendix had grossly perforated, a mortality for this group of 9.52 per cent. The mortality in the 10 cases carefully selected which had general peritonitis on admission was 60 per cent. These figures were compared with some other series recently reported in the literature.

As an aid to diagnosis, certain predisposing factors, age, sex, and seasonal incidence, were analyzed as well as the relationship of cathartic intake and postponement of treatment to development of complications and mortality. Certain symptoms and signs, including the incidence of nausea and vomiting, position of onset of pain, febrile reaction, leucocytosis, and differential count, were analyzed and although

average findings were found to be typical in each type of pathology the variability was found to be great.

Treatment was discussed, stressing the value of early operation in the nonperforated cases and early perforated cases but the advantages of delayed operation in late perforated and diffuse peritonitis groups, as well as in cases of localized appendiceal abscess. Drainage of cases and enterostomy as a complementary and secondary procedure in selected cases were analyzed. It may be stated here in relation to treatment that almost without exception the McBurney incision was used as operative approach in these cases.

Finally, associated conditions and the complications occurring postoperatively were listed and discussed.

CONCLUSIONS

1. The age group in which appendicitis most frequently occurs is in the five-year period from 16 to 20 years of age. It may occur, however, at any age and the incidence of complications, i.e., gangrene and perforation, is higher proportionately in the ages before 6 and after 50 years.

2. The male is more frequently afflicted and is more likely to have the infection go on to complication than the female.

3. Some seasonal variation in the incidence of appendicitis exists. The incidence is about 15 per cent greater in the spring and summer months than in the fall and winter months.

4. The principal predisposing factors to complications of acute appendicitis are the administration of laxatives and delay time between onset of disease and surgical treatment.

5. The symptoms and signs of appendicitis, although following average trends in relation to the severity of the pathology present, vary markedly in cases with the same pathology present. We believe tenderness over the appendix to be the most reliable sign.

6. Mortality of this series was analyzed and found to compare favorably with other series recently reported.

7. We believe our favorable mortality to be due to early emergency treatment of all early cases of acute appendicitis with conservative treatment of localizing abscesses and certain severely ill late cases of perforation with local or diffuse peritonitis. We believe the use of the McBurney incision is of value in lowering mortality because of the accessibility of the appendix and diminished trauma to bowel and unlikelihood of spreading infection when perforation has already occurred. The close coordination in this clinic between the intern, fellow, and full-time senior staffs in carrying out preoperative, operative, and postoperative treatment has certainly been a factor in maintaining a favorable mortality rate.

8. The use of drainage material is decreasing on our service although drainage is still used in many cases of perforation and, of course, in treatment of appendiceal abscess. Drainage is being used infrequently in gangrenous nonperforated cases even when cloudy fluid is present.

9. Enterostomy is still a useful operation in some badly distended patients who do not respond to Wangensteen deflation.

10. Diffuse peritonitis is the usual cause of death in acute appendicitis.

11. An appeal is made to other groups to analyze their cases in respect to the pathology present, since group mortality figures mean little and it is only by such a "break-down" analysis that one can arrive at any conclusions as to the value of any method of treatment in the cases which have progressed to perforation with its attendant high mortality.

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NONDRAINAGE IN COMPLICATED APPENDICITIS

Review of 109 Consecutive Cases

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A GENERAL review of the literature has convinced me that no dogmatic statement on the question of drainage in appendicitis can be made by a study of the literature. The old dictum, "If you are in doubt, drain," seems more and more in the past few years to be replaced in the minds of some surgeons by the contrary principle: "If in doubt, don't drain." Having this in mind, I wish to contribute my results with nondrainage operative treatment in a consecutive group of 109 cases of appendicitis complicated with peritonitis or rupture. I hope that in the future many such contributions may crystallize a uniform opinion on this controversial subject.

MORTALITY OF APPENDICITIS

The average mortality for appendicitis of all kinds from American and European clinics is about 5 per cent.[1] It is well known to all surgeons that the so-called "clean" cases of acute appendicitis have a negligible mortality, but the more serious cases, or the old so-called "drainage group" where the pathology no longer remains within the peritoneal coat of the appendix,[2] according to most writers on the subject, carry a mortality of from 10 to 25 per cent or more, excluding the cases of localized abscess.

Guthrie[2] in an editorial says our attention should be only on those cases of appendicitis in which the pathology no longer remains within the appendix and terms those cases "appendicitis with complications." He ventures the opinion that the conservative measures of treatment should show a far lower mortality, but he does not state exactly what he considers the conservative measures to be.

Kehl and Rentschler[3] in their series of 126 such serious cases had a mortality of 12.6 per cent. They practically routinely used drainage.

Haggard[4] in 379 cases of ruptured appendices reports a mortality of 5 per cent and in 186 cases of generalized spreading peritonitis with gangrenous perforation, 24.7 per cent. In 1,650 less acute cases his mortality was 0.72 per cent. He quotes other authors with somewhat the same figures on mortality.

NONDRAINAGE TREATMENT AND MORTALITY

Spivack[5] states that some surgeons, notably, Carl A. Meyer, of Chicago, in gangrenous appendicitis or if free pus and fluid have been

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found in the abdominal cavity, close the peritoneum by suture and then drain the rest of the anterior abdominal wall or close it completely, depending on the gravity of the infection. Their opinion is that the peritoneum will take care of the infection and if it cannot take care of it itself no drains will save the patient.

Thorek[6] states that at the Cook County Hospital in Chicago some surgeons of the staff often omit drainage and obtain excellent results.

Willis and Mora[7] in a study of 100 ruptured or acute suppurative cases of appendicitis with only one fatality and a study of the literature conclude: "Drainage after operation for ruptured or gangrenous appendicitis does not subtract from the mortality but does add to the morbidity." Their own study of 100 cases demonstrates the comparative safety of closing the abdomen without drainage following operation for acute suppurative appendicitis.

Potter[8] believes a lower mortality in acute diffuse peritonitis following acute appendicitis is favored by employment of suitable means to prevent ileus, for which he recommends pitressin; but he also emphasizes early operation with removal of the appendix, minimal drainage of the peritoneal cavity, and adequate amounts of water, salt, and glucose to supply fluid and chemical deficiencies.

In the *1936 Yearbook of General Surgery*[9] it is stated that at Utrecht in Holland the less frequent use of drainage in acute appendicitis does not appear to have had unfavorable influence on mortality and on the other hand has shortened the postoperative hospital stay. But, it is also stated that the decreased mortality is due to earlier operation.

In the *1937 Yearbook of General Surgery*[10] Cafritz advises nondrainage in appendiceal peritonitis and presents twenty-five cases in which nondrainage was successfully carried out.

Rhodes[1] in reviewing 1,000 consecutive cases of acute appendicitis at a San Francisco Hospital gives figures showing decidedly less mortality in cases nondrained as compared with those drained. In 32 cases of gangrenous nonperforated appendicitis that were drained, the mortality was 9 per cent and in 224 later cases of the same kind not drained the mortality was 0.9 per cent. In 89 gangrenous perforated cases that were drained the mortality was 10 per cent, while in 113 later cases of the same kind that were not drained the mortality was only 2.4 per cent. They state that for several years they have averted all intra-abdominal cases drained in peritonitis. Perforated ulcers, traumatic injuries of the gastrointestinal and genitourinary tracts are closed without drains. It is only in the chronic, well walled-off abscesses that they still use drains. They state the case against drainage in peritonitis as follows:

"Within a few hours the drain ceases to act other than as a foreign body which is walled off by adhesions.

"A foreign body, such as a drain, has been shown to lower the natural immunity of the peritoneum against infection. (This would indicate that drainage is a menace rather than a help.)

"Drainage causes temporary loss of the normal defensive peritoneal fluid.

"There is a longer and more stormy convalescence because of the added pathologic changes caused by the foreign body, resulting in partial obstruction, ileus and increased peritonitis.

"An intra-abdominal drain through an infected wound attracts and harbors all the dependent suppuration of that wound. The profuse purulent discharge from such a drained wound arises primarily from suppuration in the wall and not from within the cavity. The depth of such a drainage tract therefore collects suppuration from the abdominal wound, and hence the intra-abdominal infection surrounding the drain is aggravated.

"There is a high incidence of early and late postoperative herniae.

"Necrosis from the pressure of the drain has been the cause of many fecal fistulas and fatal hemorrhages. (There was only one fecal fistula in the 1,000 cases, a case in which drainage was employed.)

"Drainage has increased the incidence of secondary intra-abdominal abscesses in the series."

REVIEW OF CASES

Tabulated as Table I are 109 consecutive cases presented by myself. These cases have been operated upon in the past 4½ years of practice. They are all of my cases in which at operation local or generalized peritonitis was found present. No cases of this classification were excluded. Excluded from this series were all "interim appendices" or appendices removed while doing some other operation, all localized appendiceal abscesses drained, and all acute appendices in which the inflammation was confined within the appendix. Three-fourths of these 109 cases were found in the age group between 15 and 40 years. There were 49 males and 65 females.

The cases are divided into three main groups. In the first group are those cases in which at operation a local peritonitis about the appendix was present as evidenced by acute inflammation of the appendix and fresh adhesions of omentum or small gut to the appendix. The appendix in such cases was partially or totally gangrenous with pus around it. This group comprises 70 cases, obviously the cases that came to operation earliest. The mortality in this group was 0. None of these cases were drained though many times at operation doctors assisting or looking on advised drainage. Only 12.9 per cent of these cases had taken purgatives.

The second group of cases totaled 30 and were those with spreading or generalized peritonitis and the appendix was invariably totally

TABLE I

109 CONSECUTIVE APPENDECTOMIES IN ACUTE APPENDICITIS CASES THAT WERE COMPLICATED WITH LOCAL OR GENERALIZED PERITONITIS

	ACUTE, PARTIALLY OR TOTALLY GANGRENOUS WITH LOCAL PERITONITIS	ACUTE GANGRENOUS WITH SPREADING OR GENERALIZED PERITONITIS	ACUTE RUPTURED CASES, PARTIALLY OR TOTALLY GANGRENOUS BEFORE RUPTURE	TOTAL
No. of cases	70	30	9	109
Deaths	0	3	1	4
Percentage of deaths	0	10%	11.1%	3.7%
Percentage that took purgative after onset of acute symptoms	12.9%	63.3%	100%	34.1%
Drained	0	1	3	4
Died that were drained	0	0	0	0
Saved by drain	0	0	1 ?	1
Died that might have been saved by drain	0	1 ?	0	1
Cases having acute intestinal obstruction on sixth day or later with operation for	1	1	0	2

gangrenous. There was a large amount of fluid or pus widespread in the abdominal cavity and large areas of the peritoneum were inflamed even as distant as the upper abdomen or pelvis. Of these cases 63.3 per cent had taken purgatives and 3, or 10 per cent, died. One case was drained and one possibly could have been saved by drainage, though I think not. All of this second group would have been drained by any surgeon believing in the efficacy of drainage at all.

Comprising the third group were 9 cases in each of which rupture had occurred before time of operation; feces or infected contents of the ruptured appendix were being poured out into the abdominal cavity. One of these patients died, giving a mortality of 11.1 per cent for this group. Three of the earlier cases in this group were drained. None of the cases drained died and one of the drained cases was considered as possibly being saved by the drain, although I do not subscribe to this view. Nine of these cases of ruptured appendix took purgatives after the onset of the acute appendicitis. The mortality for the 37 cases that took a purgative after onset of symptoms of acute appendicitis was 10.9 per cent. The combined mortality for these 109 cases was 3.7 per cent.

OPERATING METHODS USED

All of these cases, as well as all cases of appendicitis, were operated upon at once when the diagnosis of appendicitis was made.

I do not believe in the delayed treatment, especially in rural or small town practice. In all of the cases but one the appendix was removed. The abdomen was opened by a right rectus or right paramedian incision. I believe in long incisions especially in these types of appendices as they are often misplaced and are hard to find unless there is adequate exposure. Thorough abdominal exploration is impossible through a McBurney incision and any enlargements of it will injure the abdominal wall greatly. McBurney himself did not advise its use in acute cases.

The only departures from usual operative procedures in these cases were as follows:

1. First was the method of locating the appendix after the abdominal cavity was opened. The operator stood on the left side of the patient and had the assistant strongly retract the right rectus muscle with its underlying fascia and peritoneum laterally and slightly anteriorly. While the assistant did this, the operator pushed in a moist abdominal pack on top of the omentum and intestines and with this "crawled" over the intestines and omentum to the ileocecal region where he located the appendix by direct vision. In introducing this pack and "crawling" over it to the region of the appendix and then leaving it in situ until after the appendix was removed, further contamination of the abdominal cavity was averted more completely than in any other method that I have ever tried or found in the literature. This pack caught all spill or contamination. It was not removed until all operative manipulation around the appendix was over and all mopping out or suction was completed.

2. Ligation of the appendix was practiced and none in this entire series was purse-stringed or inverted. This was time saving and was never regretted. In nearly every case the ligated mesoappendix was tied to the carbolized stump of the appendix.

3. No drainage was used, with the exception of four earlier cases.

PRE- AND POSTOPERATIVE CARE

As all cases were operated upon at once after the diagnosis was made, there is little to say about preoperative care. Postoperatively, all cases were given water, salt, and glucose by hypodermoclysis or infusion and all food and liquids by mouth were withheld until it was believed the peritoneum had cared for the infection and normal peristalsis could safely be restarted. This period varied from two to ten days. Morphine was given until it was felt safe to resume peristalsis, but never less than three days. Postoperative care was as great a factor in the successful management of these cases as the operation.

COMMENTS

1. This series of cases shows the safety of closing the peritoneal cavity without drainage.

2. Postoperative care played a large part in the recovery of all of these cases.

3. The danger of taking purgatives in acute appendicitis is clearly demonstrated by these cases, which show that 100 per cent of the ruptured and 63.3 per cent of those with spreading or generalized peritonitis had been thoroughly purged after the onset of their acute appendicitis. The mortality of these "purged" cases was 10.9 per cent.

4. Complications in this series were relatively infrequent considering the seriousness of each case and were not aggravated by lack of drainage.

5. The method of introducing an abdominal pack and "crawling" over it to the appendiceal region aided in preventing further spread of the spill from the infected focus of the appendix during both exploration and removal of the appendix.

6. A detailed study of the fatal cases found no basis for the belief that drainage or delayed operation would have saved any of them.

7. A consideration of the four drained cases showed the futility of drainage.

RESULTS

Using nondrainage treatment in all but 4 early cases in a series of 109 consecutive appendectomies complicated by local or generalized peritonitis, a mortality of 3.7 per cent was encountered. These figures compare favorably with most reports in the literature.

CONCLUSION

Nondrainage treatment of peritonitis from gangrenous or ruptured appendicitis can be concluded as safe and as a factor in reducing both mortality and morbidity.

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INTERNAL BILIARY FISTULAS

A Discussion of Internal Biliary Fistulas Based on 29 Cases

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APPROXIMATELY 1.2 per cent of all the patients with cholecystitis admitted to the University Hospitals since 1915 have had internal biliary fistulas. Such a complication should be suspected in every patient suffering from a long-standing biliary tract disease. In a series of 10,866 routine necropsies Roth, Schroeder, and Schloth[2] found 43 biliary fistulas.

Table I includes practically all types of internal biliary fistulas that have been reported in the medical literature.

Table I

1.	Duodenal
	a. Cholecystoduodenal
	b. Choledochoduodenal
	c. From cystic duct to duodenum
	d. From reformed gall bladder (dilated stump of cystic duct) to duodenum
2.	Colic, cholecystocolic
3.	Gastric
	a. Cholecystogastric
	b. Choledochogastric
4.	Jejunal, cholecystojejunal
5.	Mixed, from biliary tract to two or more viscera
6.	Bronchial
	a. Cholecystobronchial
	b. Choledochobronchial
	c. Hepatobronchial
7.	From biliary tract to kidney pelvis or ureter
8.	From biliary tract to urinary bladder
9.	From biliary tract to uterus or vagina
10.	From biliary tract to pleura
11.	Between the bile ducts
12.	From biliary tract to portal vein
13.	From biliary tract to hepatic artery
14.	From biliary tract to pericardium
15.	From biliary tract to an ovarian cyst

The varieties listed under Types 1 to 6 are given in the order of their relative frequency and importance. Bronchobiliary fistulas are rather uncommon in the United States, but they assume greater importance in the tropical countries where liver abscesses are developed more frequently. Divisions 7 through 15 include rare and relatively unimportant types of fistulas. It is, however, entertaining and somewhat startling for even the medical man to learn that gallstones have been found plugging the portal vein or hepatic artery, that both men

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and women have passed gallstones in the urine, that biliary calculi have been found with the placental membranes following labor, or that the pleural and pericardial sacs have been invaded and forced to retain the contents of a diseased gall bladder. If space permitted, a discussion of the pathogenesis[11, 12] of only these rarer forms would give one a better understanding of the other varieties.

This study is based on 29 cases from the University Hospitals. It includes 24 cholecystoduodenal fistulas; 1 cholecystogastric; 1 choledochoduodenal; 1 cholecystocolic; 1 fistula from a hepatic duct to the duodenum; and 1 from a reformed gall bladder to the duodenum. The latter 2 fistulas are apparently unique since no other similar cases were encountered in the literature. Twenty-seven of the patients were females. The average age for the group was 58 years, the youngest being 25 and the oldest 83 years.

Cholecystoduodenal fistulas are the most common type. Naunyn[3] reported 193 cases of which 93 connected the gall bladder and duodenum, 49 led to the colon, and 15 led to the lower end of the stomach. Judd and Burden[1] collected 153 cases of which 117 were duodenal, 26 were colic, and 6 were gastric. Naunyn[3] also reported 15 cases of choledochoduodenal fistulas and 2 cholecystojejunal fistulas. Fistulas between the common duct and duodenum are probably more common than is generally supposed.

PATHOGENESIS

Except for those fistulas established surgically for the purpose of obviating persistent biliary obstruction, all internal biliary fistulas are of spontaneous origin. Often the pathogenesis is as follows: After repeated attacks of acute inflammation, the gall bladder or bile ducts become adherent to the nearest portion of gut. The first part of the duodenum, the proximal end of the transverse colon, and the pyloric end of the stomach are the usual sites of involvement in order of their frequency. Then during an acute attack perforation of the adherent visceral walls occurs due to the necrotizing action of contained calculi or bulging empyema. The fistulous tract is then established, and the septic contents of the biliary tract are liberated into and carried away by the gastrointestinal tract. Another possibility is that rupture of a diseased gall bladder produces a pericholecystic abscess which secondarily necrotizes the gut wall to establish the fistula.

In all probability stones are essential in the formation of all spontaneous internal biliary fistulas except the more uncommon cases due to carcinoma in the biliary tract or to perforating peptic ulcers. Fifteen cases in this series were proved to have cholecystolithiasis; 10 had stones in the common duct; and 3 had bile sand in the common duct. Stones were found in transit in 2 fistulas.

SYMPTOMS, SIGNS, AND DIAGNOSIS

The average history in this series revealed repeated attacks of biliary tract disease. The longer the trouble had persisted, the more likelihood of there being a fistula present. One patient complained of recurring episodes for 30 years, and several had attacks for over 20 years. The average duration of the biliary tract complaints was 12 years.

The symptoms, which often included colic, fever, dull pain, dyspepsia, nausea, vomiting, intolerance to fatty foods, constipation, diarrhea, or jaundice, were essentially those associated with nonfistulous biliary tract disease. Fever, which is supposedly more marked with fistulas, was an inconstant finding. If any variation is noted, it is that with fistulas the attacks were usually more severe and gradually increased in duration and intensity. The patients frequently suffered the most severe and prolonged attack just before entering the hospital.

The textbooks on biliary tract diseases[5-7] indicate that many patients receive sudden, prolonged, or even permanent relief from all symptoms when the fistulas are formed and the septic biliary contents are liberated into the gastrointestinal tract. Only one patient in this series reported temporary relief when the fistula was formed, but she developed an acute intestinal obstruction due to gallstones within forty-eight hours. No history of prolonged or permanent relief was found.

The vomiting of gallstones is supposedly pathognomonic of the formation of a fistula between the biliary tract and the stomach. It is also said that pus and blood in the vomitus may indicate the formation of a fistula to either stomach or duodenum. The colic type of fistula is thought to produce diarrhea or to cause peculiarly colored stools which contain unevenly mixed and unoxidized bile. None of the above findings, however, were elicited in this series.

Intestinal obstruction due to gallstones that have passed through fistulas is reported in the textbooks[5-7] and literature[1, 13] as a rare secondary complication. In this series, however, 9 cases of intestinal obstruction were discovered. The calculi were impacted somewhere along the small bowel in 7 cases, and in 2 cases the obstruction was due to inflammatory adhesions about the fistulas which contained calculi. The diagnosis of acute intestinal obstruction due to gallstones should be considered in any patient who suddenly develops obstructive signs and symptoms after a prolonged siege of biliary tract disease.

The passage of gallstones with the feces is always suggestive of an internal biliary fistula, but the stones are definitely significant only if they are too large to have passed through the ampulla of Vater. One of three patients who were diagnosed preoperatively in this series passed small gallstones rectally on several occasions, but during one defecation she passed such a large calculus that she fainted during its painful delivery.

The second patient diagnosed preoperatively had developed a tender palpable gall bladder during a typical attack of acute cholecystitis. After a few days the palpable mass disappeared together with the associated acute pain. Within the next forty-eight hours the patient developed an acute intestinal obstruction and at operation a large gallstone was found impacted in the lower ileum. This patient is also the only one in whom the symptoms indicate the time of formation of the fistula.

The third case diagnosed clinically was the cholecystocolic fistula which was found during a routine roentgenologic examination of the colon. The barium mixture passed through the fistula to fill the gall bladder and biliary radicles which were then easily visible on the roentgenogram. Roentgenologic studies are only rarely of aid in the diagnosis of internal fistulas. One interesting case report by Vorhaus and Rogers[4] describes a cholecystocolic fistula which was accidentally visualized roentgenologically by means of gas which passed from the colon through it to fill the biliary radicles.

The diagnosis of an internal biliary fistula is rarely made during the clinical examination, and the fistula is usually an accidental finding at the operating table. Even then it may be missed, as in two patients who were thought to have carcinomas after large ligneous masses had been found at operation. The post-mortem examinations in both cases, however, revealed the carcinomatous-like masses to be composed of dense cicatricial tissue surrounding fistulous tracts and calculi. Twenty-six of the fistulas in this series were not diagnosed as such until after operation or post-mortem examination.

PROPHYLAXIS

What can be done to prevent the formation of internal biliary fistulas? At present the best prophylactic measure is the removal of the gall bladder and all calculi in the early stages of the biliary tract disease. An obstruction in the biliary channels is without doubt a dominant factor in the production and maintenance of a fistula. Relief of this obstruction in most cases would allow the tract to heal spontaneously. Moreover, the tendency for the fistula to be healed or obliterated by cicatricial tissue is so great that it practically never serves any length of time as an adequate channel for the flow of bile and therefore never gives prolonged relief from the biliary tract symptoms. For that reason it is imperative to relieve the biliary tract pathology before the fistula develops.

OPERATIVE FINDINGS AND DIFFICULTIES

After the fistula has been established, the difficulties of the operative procedures are greatly increased. The surgeon frequently finds the viscera in the right upper abdomen to be so closely matted by inflam-

matory adhesions that no line of cleavage is discernible, and adequate exposure is obtained only by blind probing and cutting. The walls of the gall bladder and bile ducts are usually thickened and fibrotic from repeated attacks of inflammation. Previous operative procedures merely add to the amount of scarring and fibrosis. Not uncommonly the adhesions in the pericholecystic area are so dense and massive that drainage of the gall bladder is all that dare be attempted.

If the surgeon is to cure the patient, however, he is confronted not only with the necessity of relieving the original biliary obstruction, but in addition he is forced to excise the fistula and then close the defect which remains in the involved portion of gut wall.

Sometimes the fistula is nothing more than a side-to-side anastomosis between the affected viscera. Again it may be several centimeters long running through heavy cicatricial tissue and containing much necrotic and septic debris in its lumen which may be pinhole in size or large enough to allow the passage of a large stone. When the lumen is small, the surgeon may excise the fistula and inadvertently overlook the opening in the gut wall, with the result that peritonitis, subphrenic abscess, pelvic abscess, or even death supervenes. If the fistulous lumen is large, the cavity must often be drained like any other intra-abdominal abscess.

The defect in the gut wall may be surrounded by friable scar tissue which makes the repair very difficult and which makes the possibility of a permanent closure a questionable one. Most of the fistulas lead to the duodenum which is noted for its resistance to successful closure. If the defect is successfully sutured or a purse-string inversion of the ligated fistulous tract is obtained, a stricture of the gut wall may result. A gastroenterostomy or some other shunting operation is then required. The above difficulties are further increased if the fistulas are multiple or enter more than one division of the gastrointestinal tract.

If intestinal obstruction has developed due to calculi, strictures, or hypertrophic mucosa, the entire surgical attack sometimes must be directed toward relieving the bowel obstruction. The fistulous tract is then left intact with the hopeful prayer that the patient will not develop biliary obstruction, suppurative cholangitis, or greater liver damage before the gastrointestinal condition has subsided.

Associated pathologic findings may include acute or chronic hepatitis and pancreatitis. Both of these conditions are undoubtedly present more frequently than recognized, and they greatly increase the patient's as well as the surgeon's difficulties. Liver damage in 3 cases which came to autopsy was reported as acute hepatitis, occlusive biliary cirrhosis, and suppurative cholangitis respectively. Pancreatitis was noted in 10 cases.

Unfortunately the patients in this series did not receive adequate prophylactic care. Although the average duration of symptoms was 12 years, 24 of the 29 cases had had no previous surgical procedures on their biliary systems. Three of the previously operated cases had had cholecystostomies, and only 2 had had cholecystectomies. One of the cholecystectomy cases had a fistula from a hepatic duct to the duodenum and the other had a fistula from a re-formed gall bladder to the duodenum.

OPERATIVE PROCEDURES AND RESULTS

Twenty-five of the patients were treated at the University Hospitals by operative procedures which included 12 cholecystectomies, 1 cholecystostomy, 9 choledochostomies, 1 transplantation of the common duct, 12 duodenal repairs, 2 gastric repairs, 3 gastroenterostomies, 4 enterostomies for removal of stones, 1 laparotomy for the crushing of a gallstone in the lower ileum, and other lesser procedures. Two patients died as a result of the fistulas and related complications before surgery could be instituted. Two patients refused surgery. Twelve of the patients receiving surgical treatment were relieved by the operative procedures, although the 1 patient treated by cholecystostomy developed a persistent external biliary fistula. Thirteen of the cases died following the surgical procedures.

MORTALITIES

The following brief analysis of the mortalities emphasizes the complications and difficulties that may arise in the treatment of these patients.

Five cases died as a result of intestinal obstruction due to biliary calculi. Two of these expired even before surgery was instituted. A sixth death due to intestinal obstruction resulted from marked scarring and contraction about a cholecystoduodenal fistula and several calculi.

In 3 cases peritonitis following the difficult operative procedures was the main cause of exodus. One of these was complicated by acute hepatitis and nephritis, another had an impacted common duct stone, and the third had bronchopneumonia.

One patient died after many repairs of the common duct and one choledochoduodenal intubation had been carried out. The autopsy revealed suppurative cholangitis, stenosis of the common duct, an external biliary fistula, a fecal fistula from the colon, and an occlusive biliary cirrhosis of the left lobe.

In 3 deaths the causes were varied: first, postoperative shock and septicemia; second, subphrenic abscess; third, partial obstruction of the common duct and bronchopneumonia.

Death in 2 cases was not related to the disease of the biliary tract since the autopsies revealed a carcinoma of the cecum in 1 patient and an infected decubitus ulcer with septicemia in the other.

Due to the fact that these patients present such a myriad of possibilities, individual study of each case is necessary for a thorough explanation of the final outcome. It would seem from this study, however, that delay and lack of prophylactic treatment were essentially responsible for 13 of the deaths, 2 deaths being unrelated to the biliary tract disease. The 3 cases of peritonitis, the subphrenic abscess, and the case of postoperative shock and septicemia were definite sequelae of the surgical procedures. In the remaining 8 deaths, however, the dominant factor is the persistence of untreated biliary tract disease.

CONCLUSIONS

1. The formation of a communication between the biliary tract and gut is sometimes a life-saving accident because it allows inflammatory debris and calculi to be spontaneously extruded from the biliary system and to be carried away with the feces.

2. Nevertheless, it breeds possibilities for greater liver damage by providing an open channel through which septic intestinal contents are flushed into the biliary radicles.

3. It rarely provides adequate biliary drainage because scar formation and contracture of the fistulous lumen begin almost as soon as the communication is established. Future biliary obstruction is therefore inevitable unless curative surgery is instituted.

4. It usually occurs after a prolonged history of biliary tract disease which has gradually been increasing in severity and which most likely has never been approached surgically.

5. It fosters the development of various types of intestinal obstruction arising from impacted gallstones, scarring of the gut walls, inflammatory adhesions, or hypertrophic mucosa.

6. It impedes accurate diagnosis and, although its presence should always be suspected in cases of long-standing biliary tract disease, it usually cannot be proved preoperatively.

7. It increases the risk of surgical treatment many times over that of uncomplicated biliary tract disease which has an operative mortality of 1 to 5 per cent.

8. It greatly increases the nonoperative mortality and morbidity of biliary tract disease.

9. The bulk of evidence indicates that practically all internal fistula to the gastrointestinal tract can be prevented by early surgical treatment of the predisposing chronic cholecystitis or related biliary tract disease and that delay should be strenuously avoided.

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BACTERIAL AGGLUTININ IN BILE OF PATIENTS WITH CHOLELITHIASIS

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THE relation of infection and cholelithiasis has received considerable attention, although a specific mechanism which is responsible for gallstone formation has not yet been described. It has recently been demonstrated that the gall-bladder wall, when damaged by infection, permits bile salts to be rapidly absorbed from the gall-bladder bile[1] and it has also been shown that hepatitis results in a bile in which the bile salt concentration is greatly reduced. A reduction in the bile salt concentration may permit cholesterol to precipitate out of the bile. This mechanism is perhaps an important one in gallstone formation but it is quite possible that other factors also may be involved. I have found that agglutinins appear in the bile of dogs in response to a specific infection.[2, 3] The presence of antibody in bile may be a significant factor in stone formation because of its ability to cause agglutination or precipitation, thus providing a nidus for stone growth. The present study demonstrates that antibody is present in human bile and that the agglutinin content of human bile varies in individuals with disease of the biliary apparatus being greatest in patients with cholelithiasis.

METHOD

Material for this study was obtained from patients on the surgical services of Dr. I. S. Ravdin and Dr. E. L. Eliason in the Hospital of the University of Pennsylvania. After exposing the gall bladder, its contents were aspirated and the bile was placed in a sterile container. In those patients in whom drainage of the common duct was instituted, samples of hepatic bile were obtained for study. All specimens removed at the operating table, and subsequent to operation, were collected under aseptic precautions. In the majority of cases within forty-eight hours of operation blood from the antecubital vein was obtained for serum agglutinin titer and blood culture.

One-half cubic centimeter of bile was inoculated into glucose-beef and plain beef-infusion broth for study under aerobic and anaerobic conditions. Samples of bile and serum were studied for the presence of agglutinin to *Staphylococcus aureus*, *B. coli*, and *B. typhosus*.

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The agglutination tests were performed by serial arithmetic dilution of the serum and bile to a volume of 0.8 c.c. with isotonic buffered phosphate solution (pH 7.4) which contained 0.014 thymol. Bacterial antigen was added so that the total volume was 1 c.c. The agglutination test tubes were incubated at 37° C. for twenty-four hours and were then examined with a hand lens for evidence of agglutination. Where there was any doubt about agglutination, the test tubes were centrifuged and the sediment was resuspended in 1 c.c. of physiologic saline solution, after which they were examined again for evidence of agglutination. Simultaneous controls of sera, bile, and all antigens were included with each test. The original strains of bacteria were obtained through the courtesy of Dr. Stuart Mudd, of the Department of Bacteriology of the University of Pennsylvania.

The antigens used were *Staphylococcus aureus* (hemolytic), *B. coli*, and *B. typhosus*. These organisms were kept in stock culture on agar slants and in beef-infusion broth (pH 7.4). Stock cultures were transferred every five to six weeks. Antigens were prepared from 250 c.c. of beef infusion broth in which the organisms had been cultured for twenty-four hours at 37° C. After centrifuging, the supernatant fluid was decanted and the bacterial residue was washed twice with physiologic salt solution and was then resuspended in 25 per cent of the original volume with normal saline solution. The bacteria were killed with thymol (0.1 per cent). After twenty-four hours in the refrigerator the bacterial antigen was cultured for sterility and a smear was performed to check the bacterial content. The antigens were diluted to contain four to five billion organisms per cubic centimeter. Antigens were discarded if and when spontaneous agglutination or contamination occurred. No antigen was used for more than fifteen days. A Gram stain was performed daily on all antigens and cultures in use.

RESULTS

There were two main groups among the 23 patients from whom bile specimens were obtained. Sixteen patients had cholelithiasis; 7 did not. The latter group of patients were operated upon for chronic cholecystitis (3), carcinoma of the pancreas (2), icterohemolytic anemia (1), and congenital atresia of the common duct (1). Agglutinin to staphylococci, *B. coli*, and *B. typhosus* was present in the serum of all but one of the noncalculous patients, yet in no patient was the agglutinin titer greater than 1:64. Hepatic bile was obtained from 3 patients and no agglutinin was present in these specimens. Gallbladder bile was secured from 6 of these patients. Only one of these contained agglutinin and this was to the colon bacillus (1:8) (Table I).

Specimens of bile were obtained from 16 patients who had cholelithiasis. Some of these had mixed, faceted stones, while others had

TABLE I

AGGLUTININ TITER IN PATIENTS AT OPERATION

NO GALLSTONES PRESENT

CASE NUMBER	SERUM			GALL-BLADDER BILE			HEPATIC BILE		
	A	C	T	A	C	T	A	C	T
28729	0	0	0	0	0	0	–	–	–
31940	32	8	16	0	0	0	0	0	0
32105	12	6	6	–	–	–	0	0	0
32266	32	2	0	0	0	0	–	–	–
32637	64	0	32	0	0	0	0	0	0
32725	40	10	10	0	0	0	–	–	–
32766	64	64	16	0	8	0	–	–	–

single or multiple cholesterin stones. Agglutinin in significant titer was present in the hepatic bile of 2 of 6 patients. In each of the 12 samples of gall-bladder bile which were obtained, agglutinin to at least one bacterial antigen was present in titer of 1:16 or greater. In 11 blood specimens which were studied, the serum agglutinin was found to be of high titer (Table II).

TABLE II

AGGLUTININ TITER IN PATIENTS AT OPERATION

GALLSTONES PRESENT

CASE NUMBER	SERUM			GALL-BLADDER BILE			HEPATIC BILE		
	A	C	T	A	C	T	A	C	T
29135	160	80	160	128	64	128	–	–	–
31404	–	–	–	–	–	–	8	4	0
31633	320	0	20	64	0	16	–	–	–
31817	12,800	200	1,600	128	0	16	–	–	–
31848	–	–	–	–	–	–	0	0	0
31893	–	–	–	–	–	–	8	0	16
31897	–	–	–	–	–	–	0	0	0
31943	100	0	0	128	0	0	0	0	0
31950	160	20	20	12	6	0	–	–	–
32209	320	0	0	32	0	0	–	–	–
32401	1,280	40	40	32	0	0	–	–	–
32527	128	10	32	20	0	0	0	0	0
32552	1,024	64	16	64	8	0	–	–	–
32558	64	16	64	8	16	2	–	–	–
32574	64	32	32	4	16	8	–	–	–
32592	–	–	–	64	0	0	–	–	–

DISCUSSION

These studies are reported with the suggestion that several factors may be responsible for the formation of gallstones. They demonstrate that agglutinin is present in the bile of patients with cholelithiasis, even when almost pure cholesterin stones are found, and that agglutinin is not as a rule found in the bile of patients who do not have gallstones and who are thought to have had chronic cholecystitis.

The presence of agglutinin indicates the pre-existence of infection either in the gall bladder or elsewhere. The action of agglutinin in contact with its specific antigen may produce, as antigen-antibody

complex, a nidus which in itself may be a calculus or which may provide a nucleus for stone formation, particularly when the physiochemical equilibrium of the biliary constituents has been altered by disease in either the liver or gall bladder. Stone growth is more apt to occur in the presence of stasis, and such a condition more frequently occurs in the gall bladder. Although stasis is, of course, present in marked degree in carcinoma of the head of the pancreas, stone formation in this condition is rare, even though the bile salt concentration of liver from patients with this lesion may be greatly reduced. However, agglutinins were not found in the bile of the patients studied with this disease. Thus, it may well be that the coincidence of two or more factors acting in association with each other is necessary before stone formation actually takes place.

SUMMARY

1. Agglutinin titer for the staphylococcus, *B. coli*, and *B. typhosus* was determined in the serum, gall-bladder bile, and hepatic bile obtained at operation from a series of 23 patients.

2. In the specimens obtained from the patients who did not have gallstones, no agglutinin was present in hepatic bile; and agglutinin was present only to the colon bacillus in the gall-bladder bile of 1 of 6 patients. The serum of these patients contained bacterial agglutinin in low titer.

3. The hepatic bile in the patients with cholelithiasis contained agglutinin in 2 of 6 instances. The gall-bladder bile contained agglutinin in every one of the 12 patients with calculi. The serum agglutinin titer in these patients with gallstones was high.

4. The presence of bacterial agglutinin in patients who have cholelithiasis and the absence of bacterial agglutinin in those patients who do not have biliary calculi may be of significance in the etiology of gallstones.

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NONPARASITIC SOLITARY CYST OF THE LIVER*

Treatment with Carnoy's Solution

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NONPARASITIC solitary cysts of the liver are relatively rare, no more than 230 cases having been reported in the literature. It has been suggested that such cysts represent a part of the condition known as polycystic disease; however, this opinion is in the minority. The consensus is that these cysts are congenital in nature and are of biliary origin, perhaps arising from aberrant bile ducts, resulting in benign cystic adenomas.[1] The cysts are thin walled and contain a thin, watery liquid which, characteristically, is not bile stained. They vary greatly in size, as might be expected, the largest yet reported being the one described in this report. Microscopically, the walls consist of three layers: an outer layer of loose-meshed connective tissue, moderately vascularized; a middle layer of relatively dense fibrous tissue; and an inner layer of a single thickness of low columnar or cuboidal cells.

Clinically, the symptoms are variable, there being but one constant finding; that is, the presence of a mass. Only one case has been correctly diagnosed before operation.[2] The common preoperative diagnoses have been hydrops of the gall bladder, chronic cholecystitis, hydronephrosis, mesenteric cyst, ovarian cyst, and tuberculous peritonitis.[1]

Most of the cysts have been located on the under surface of the liver and in the neighborhood of the cleft between the lobes. There is nothing constant about the location, however. The disease may appear at any age, the most common period being between the ages of 40 and 60 years. Females are affected more often than males, in the ratio of 4 to 1.[3]

Treatment has been by excision, although successful results have been reported following simple drainage and marsupialization.

The chief operative complications have been hemorrhage and shock and the mortality has been rather high. Winkle and Charache,[1] in 1936, reported a mortality rate varying from 12 to 32.3 per cent in different series of cases and C. R. Davis,[4] in 1937, found reports of 50 deaths in 226 operations, a rate of 27.2 per cent.

I am reporting this case of nonparasitic solitary cyst of the liver because the condition is relatively rare, because of the unusually large size of the cyst and its unusual location, and because of the use of a sclerosing solution (Carnoy's solution[5]) as a means of obliterating it; this solution has been used for the obliteration of brain cysts, thyroglossal duct cysts, pilonidal cysts, and brachial cysts, but, so far as I know, it has not been used before in intraabdominal cysts.

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CASE REPORT.—This patient, a white female, 50 years of age, stated that, while working on a rug on June 15, 1938, she suddenly experienced a sharp, shooting pain in the region of her right clavicle. The pain radiated up the side of her neck to her head and was fairly constant but did not interfere with her sleep. She had considerable anorexia thereafter and restricted her meals to liquids on many occasions. There was no vomiting. One week later pain developed in the region of the xiphoid; this was similar to the pain in the shoulder region but more severe; it did not radiate and was not influenced by meals or by the type of food; this pain was felt more on deep inspiration. There was no coughing or spitting of blood. Some relief was obtained by strapping the chest. The diagnosis of pleurisy with gallstones was made and later another physician suggested neuritis. Three weeks after the first onset of pain, she was given x-ray treatments to the right costal margin for the neuritis, which caused considerable nausea and vomiting. About this time, she noticed a swelling in the abdomen in the region of the xiphoid.

She had never become jaundiced and her bowels had been regular up to a few days preceding the x-ray treatments. There was no fever and no chills. Past and family histories were irrelevant.

Examination at St. Luke's Hospital about four weeks after the onset of symptoms showed the following positive conditions: A mass in the right hypochondrium, with very slight tenderness. It was noted that the patient became nauseated and had severe pain in the back when lying on her back. On July 7 a gastrointestinal series and gall-bladder dye study were made. A partial quotation from the report of this examination follows: "... A complete examination of the gastrointestinal tract shows no evidence of pathology except a gall-bladder seat in the cap, a little four-hour retention in the stomach, and a definite appendix. The gall bladder does not fill at any time. This is undoubtedly a pathologic gall bladder with cystic duct obstruction."

Blood examination revealed nothing of significance beyond a slight anemia. The urine was normal.

The intern who made the physical examination at St. Luke's Hospital records his impressions as follows:

"(1) Pathology in the lower dorsal or upper lumbar spine?

"(2) Mass in the epigastrium?

"(3) Findings and history do not correlate very well for me. Have considered gastric carcinoma, ulcer, ventral hernia, gall-bladder disease, arthritis, mediastinal lesion (nerve injury) but I am unable to reach any conclusion."

A tentative diagnosis of gall-bladder disease was made and I was asked to see the patient. I examined her on July 11, 1938. At that time, the upper margin of liver dullness was at the third interspace anteriorly. The liver margin appeared to be one hand's breadth below the rib margin. In the area between the anterior ends of the sixth, seventh, and eighth ribs and the xiphoid was a bulging which fluctuated. I advised exploration with a probable diagnosis of subdiaphragmatic accumulation, probably secondary to gall-bladder disease.

Operation was performed July 13, 1938, under ethylene-ether anesthesia. The abdomen was opened by the Bevan S-shaped incision. Presenting in the wound was a tense fluctuating mass lying between the liver and the diaphragm, the upper surface of the liver being pushed down to about the level of the umbilicus. In addition to considerable spilling, 3,300 c.c. of watery fluid was withdrawn from the mass by suction and it was then found that we were dealing with a relatively thin-walled sac which apparently extended over the pericardium and great vessels medially, the diaphragm above and laterally, the liver inferiorly, and the abdominal wall anteriorly. That part of the sac anteriorly seemed quite friable. Examination of the interior of the sac visually, after inserting a light, disclosed smooth, although wrinkled, walls without nodules or local thickening and no apparent source for

the liquid. A sample of the liquid sent to the laboratory was reported negative for pus and echinococcus. A portion of the sac wall was removed for microscopic study.

In view of the friability of the sac and its widespread relations, it was felt that to attempt removal was unwise; therefore, a rubber tube was inserted into the cavity and the edges of the hole in the sac were sutured to the peritoneum. The abdomen was closed in layers.

The postoperative course was relatively uneventful.

The pathologic report by Dr. Arthur Wells, on the basis of the tissue removed, stated that the sac was lined with a single layer of columnar epithelium and there were a few small glands of uniform size in the walls with cuboidal epithelium. The walls were made up of fibrous connective tissue without inflammatory reaction. The pathologic diagnosis was cyst, probably benign, probably of bile duct origin.

The drainage from the cyst, postoperatively, was quite irregular; at times there would be no drainage for twelve to twenty-four hours and then, after the patient moved around in bed, there would be an escape of a considerable quantity of watery fluid.

About four weeks after the operation, there being no apparent tendency for the cyst to close and because of the definite epithelial lining disclosed microscopically, it was felt that, unless some means of removing or destroying the lining of the sac was found, there would be a persistent sinus. A review of the literature, abstracted in the preceding pages, disclosed a disturbingly high mortality rate following attempts to enucleate such tumors. Therefore, on Aug. 9, 1938, about 80 c.c. of sclerosing fluid, known as Carnoy's solution,[5] was introduced into the cavity through the tube, with the patient supine, and left for four minutes; then the patient was turned on her left side for one minute, and then slowly on her right side until fluid appeared at the outer end of the drainage tube. A small catheter was then introduced through the tube and the cavity thoroughly irrigated with saline solution. This procedure was carried out with no more than a very mild psychic reaction. Carnoy's solution consists of: absolute alcohol, 60; chloroform, 30; glacial acetic acid, 10; and ferric chloride, 10. There was moderate drainage following this treatment.

On Aug. 23, 1938, the capacity of the cavity was reduced to about 20 c.c. and at this time a second injection of sclerosing fluid was made, using the same technique as at the first injection. On Sept. 1, 1938, the drainage tube was removed. The patient was last seen on Nov. 8, 1938. The wound was healed; the patient had gained 22 pounds in weight and was apparently well.

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THE EFFECT OF THERMAL TRAUMA ON BLOOD VOLUME, SERUM PROTEIN, AND CERTAIN BLOOD ELECTROLYTES: AN EXPERIMENTAL STUDY OF THE EFFECT OF BURNS

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THE subject of burns has been investigated extensively from many approaches. Thus, the role played by shock,[1, 2] the bleeding volume,[3] adrenal disturbance,[4] toxins,[5-8] bacterial infection,[9, 10] fluid losses,[2, 11, 12] changes in blood electrolytes[13] and in carbohydrate and lactic acid metabolism[14, 15] has been studied. Investigations of the effect of severe burns on the circulating blood volume in relation to other organic changes have not been found in the literature.

The purpose of this study was to determine changes in the circulating blood volume, blood gases, hemodynamics, blood electrolytes, body fluids, and body temperature resulting from an extensive burn, and to correlate the behavior of the animal with functional changes in the renal, respiratory, and cardiac systems and with the pathologic findings at necropsy.

MATERIAL STUDIED

Five large male and two large female normal mongrel German shepherd dogs were used. Four of these, three males and one female, were splenectomized from fourteen to thirty-three days before the experiments. The spleens were grossly normal and recovery from operation was complete prior to the experiment. Thermal trauma was applied under anesthesia to one side of the body only in Experiments 1, 2, and 5, and to both sides in Experiments 3, 6, 7, and 8, the area involving the thorax and abdomen from the level of the clavicle to the groin and laterally to the posterior axillary line.

METHODS

Plasma and blood volumes were determined by the direct method of Gibson and Evans.[16] Colorimetric determination of the concentration of dye in serum samples was made with the spectrophotometer, quartz cells of 5 mm. depth being used, and 6 mg. of Evans blue were injected intravenously for each determination. With this method no correction for blood lost in sampling or from washing out of the can-

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nula is necessary since the measurement obtained represents the circulating blood volume at the time of dye injection.[17] The hemoglobin concentration was determined by the method of Evelyn,[18] oxygen content and CO_2 content by the gasometric method of Van Slyke and Neill,[19] serum and urine chlorides by the method of Volhard,[20] serum potassium by the method of Shohl and Bennett,[21] serum protein by the micro Kjeldahl method,[22] and urine specific gravities by a gravimetric method.

EXPERIMENTAL PROCEDURE

Animals were fasted but given water *ad libitum* for twenty-four hours before the experiment. Anesthesia was induced by the intravenous injection of sodium pentobarbital in doses of 32.5 mg. per kilogram of body weight and maintained at adequate levels through the *entire* experimental period by supplementary intravenous injections of from 32.5 mg. to 97.0 mg. of pentobarbital. Animals were weighed on a lever type platform scale sensitive to 0.1 kg. prior to anesthesia and at intervals during the procedure. A continuous record of the systolic arterial pressure was obtained on the Harvard kymograph by means of a cannula inserted into the right femoral artery. Total blood lost in washing out the cannula varied from 48 to 130 c.c., averaging 80 c.c., or about 2.5 per cent of the average total blood volume. The jugular veins were exposed for ease in blood sampling. Urine samples were obtained through an inlying catheter. After anesthesia was induced, areas of the body surface were shaved and exposed to thermal trauma by means of a Bunsen burner applied for periods of from four to twelve minutes in such a way as to produce uniform changes in the appearance of the skin throughout the area traumatized. The effectiveness of anesthesia was not altered by this procedure. Excessive heat loss by radiation from the shaved areas was prevented by covering the animal with a sheet. Rectal temperatures were taken at intervals. Arterial blood samples were taken from the left femoral artery. Tissues in the regions of the exposed vessels were kept moist with sponges wet with normal saline. Observations were made continually throughout the studies. In Experiment 1 the procedure was terminated by the intravenous injection of 170 c.c. of air and in Experiments 2, 3, and 5, of 937, 975 and 325 mg. of pentobarbital, respectively. In Experiments 6, 7, and 8 the observations were continued until the demise of the animal.

RESULTS

Changes in plasma, cell, and total blood volume, hematocrit, hemodynamics, blood gases, and electrolytes are shown in Table I. The relationship of these changes throughout the observation period in Experiment 7 is shown in Fig. 1.

PLASMA VOLUME

In all cases a decrease of plasma volume occurred. A considerable decrease, amounting to 26.6, 32.1, and 36.6 per cent, in Experiments 5, 6, and 7 respectively, followed immediately after the trauma with a continued decrease at a lesser rate during the remainder of the experiment, so that the plasma volume was reduced greatly at the end of the experimental period in all cases. The degree of reduction from initial levels was somewhat greater in the splenectomized animals than in those not splenectomized. In general, the longer the experimental period, the greater was the reduction in plasma volume.

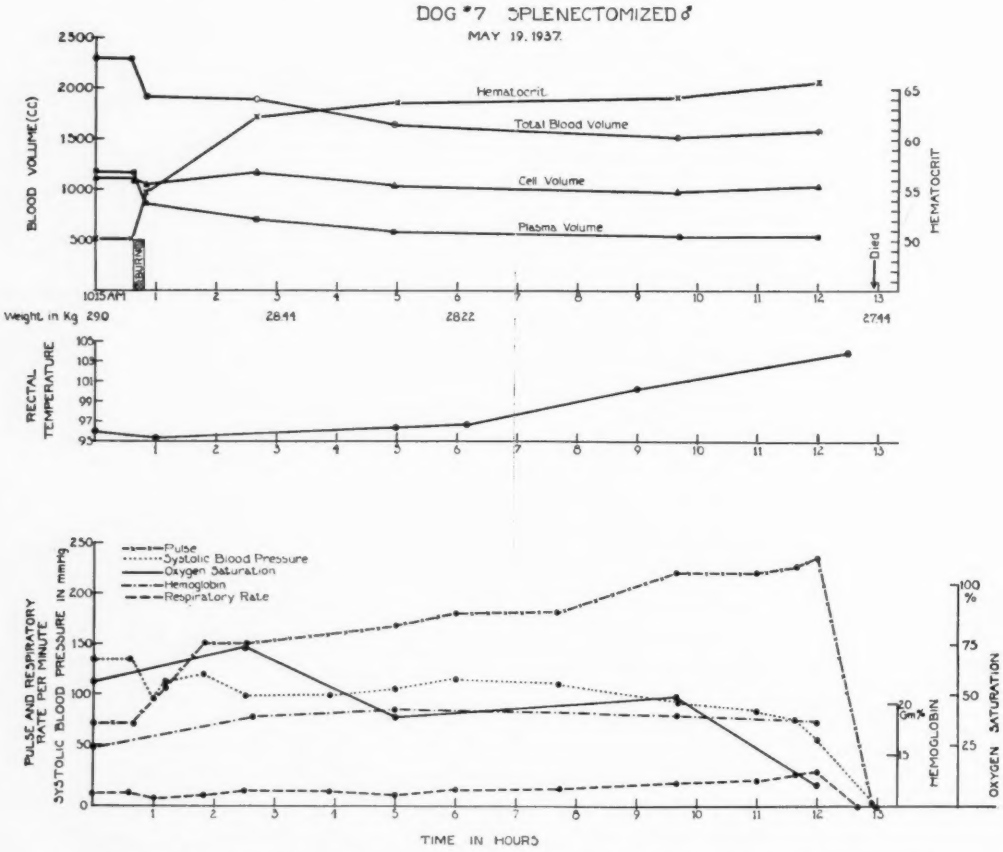


Fig. 1.—The course of changes in plasma, cell, and total blood volume, hematocrit, hemoglobin content, and oxygen saturation of venous blood; systolic blood pressure, pulse, and respiratory rates in Experiment 7.

RED CELL VOLUME

A marked contrast in the course of the circulating red cell volume was observed between the nonsplenectomized and splenectomized animals. Thus, in Experiments 1, 2, and 3 there was a marked increase in the total volume of the circulating red cells. This increase occurred

shortly following thermal trauma, and there was a general tendency for this increase to be sustained throughout the course of the experimental period. The circulating red cell volume was increased 59.5, 24.6, and 22.3 per cent above initial levels in Experiments 1, 2, and 3 respectively.

In the four splenectomized animals the circulating red cell volume decreased, there having been a reduction of 27.8, 7.8, and 13.1 per cent in Experiments 6, 7, and 8 respectively and little change in Experiment 5.

TOTAL BLOOD VOLUME

A marked difference in the course of total blood volume was observed in the nonsplenectomized and splenectomized animals due to the different response of the red cell volume, there being slight increases and only moderate decreases in the nonsplenectomized animals but considerable reductions in the splenectomized animals. The greatest reduction in total blood volume in nonsplenectomized animals occurred in Experiment 2, amounting to 20.6 per cent. The characteristic course of total blood volume in the splenectomized animals was a progressive reduction, amounting to 33.6, 41.6, and 30.8 per cent in Experiments 5, 6, and 7 respectively at the end of the experimental period.

HEMODYNAMICS

The initial pulse rate varied considerably in the different animals, but in general there was a tendency for the pulse rate to rise gradually as the experiment progressed, with a sharp rise to very high levels terminally. Initial blood pressures also varied considerably. During the course of the thermal trauma arterial pressure dropped sharply, as much as 70 mm. of mercury in Experiments 3 and 8. This fall in pressure was accompanied by a rise in pulse rate and little change in respiratory rate. Following trauma, the arterial pressure tended to return to the initial level. In the three nonsplenectomized animals the initial level was maintained throughout the experimental period. In the four splenectomized animals, following the recovery from the reduction accompanying the thermal trauma, there was a gradual and steady fall. In Experiment 5 systolic pressure was 40 mm. of mercury at the time the experiment was terminated by the intravenous injection of pentobarbital. In Experiments 6, 7, and 8 death occurred at nine, thirteen, and nine hours after thermal trauma and shortly before demise the systolic pressure fell sharply. In these three animals the terminal fall in pressure was preceded by a marked increase in the respiratory rate which was 120 per minute in Experiment 8 shortly before death.

TABLE

CHANGES IN PLASMA, CELL, AND TOTAL BLOOD VOLUME,

TIME (HR., MIN.)	WEIGHT (KG.)	RECTAL TEMPERATURE (° F.)	BLOOD VOLUME						HEMATOCRIT (VOL. %)	PULSE (PER MIN.)	SYSTOLIC BLOOD PRESSURE (MM. HG)	RESPIRATIONS (PER MIN.)
			PLASMA (C.C.)	% CHANGE	CELL (C.C.)	% CHANGE	TOTAL BLOOD (C.C.)	% CHANGE				
Dog 1.—Normal,												
Initial												
5:38 P.M.	19.65	–	710		730		1440		52.6	136	157	10
1 12	19.65	–	636	-10.4	944	+29.3	1580	+ 9.74	61.1	132	141	15
3 10	19.65	–	530	-25.4	890	+20.6	1420	- 1.4	65.1	120	145	10
5 41	19.5	–	497	-30.0	1033	+41.5	1530	+ 6.25	69.8	136	135	11
9 18	19.20	–	426	-40.5	1164	+59.5	1590	+10.4	74.2	156	143	8
Dog 2.—												
Basal												
11:09 A.M.	32.03	–	1360		1015		2375		42.9	220	100	14
1 47	31.69	100.4	1142	-16.05	1118	+10.15	2360	- 0.63	51.5	237	184	19
4 10	31.54	100.5	953	-29.9	1285	+16.75	2238	- 1.56	57.5	212	114	25
7 35	–	100.7	750	-44.8	1135	+10.84	1885	-20.6	60.2	252	90	15
10 36	30.82	102.7	715	-52.5	1365	+24.6	2080	-12.4	65.7	252	102	23
Dog 3.—												
Basal												
11:14 A.M.	26.54	97.4	1000		965		1965		49.0	144	176	20
1 11	25.84	99.8	952	- 4.8	1038	+ 7.58	2090	+ 6.37	54.5	160	190	24
5 22	–	101.0	800	-20.0	1240	+29.0	2040	+ 3.81	60.2	180	170	16
9 04	25.54	104.5	685	-21.5	1210	+22.3	1895	- 3.81	63.9	230	158	36
Dog 5.—April 27, 1937.												
Basal												
9:51 A.M.	24.44		1200		940		2140		43.8	134	125	10
1 01	–		880	-26.6	800	- 6.48	1680	-21.5	47.6	154	122	10
2 17	–		848	-29.3	947	+ 0.7	1795	-16.1	52.9	168	140	18
5 11	24.16		672	-44.0	983	+ 4.6	1655	-22.6	59.4	180	114	18
6 55	24.06		650	-45.7	935	- 0.5	1585	-25.7	59.1	170	64	10
11 33	23.86		592	-60.7	828	- 1.28	1420	-33.6	58.3	240	74	36
Dog 6.—May 10, 1937.												
Basal												
10:13 A.M.	25.8	100.6	1208		832		2040		40.8	168	120	10
0 53	25.5	100.6	820	-32.1	680	-18.60	1500	-26.4	45.5	144	128	18
3 11	25.35	100.3	695	-43.9	700	-15.9	1395	-31.6	50.2	168	128	16
6 54	25.0	102.2	658	-45.6	747	-10.2	1405	-31.1	53.5	180	72	44
8 35	24.8	102.7	590	-51.2	600	-27.8	1190	-41.6	50.4	200	40	34
8												

I

BLOOD GASES, SODIUM CHLORIDE, AND SERUM PROTEIN

OXYGEN (VOL. %)	HEMOGLOBIN (GM. %)	OXYGEN CAPACITY (VOL. %)	OXYGEN SATURATION (%)	CARBON DIOXIDE (VOL. %)	SERUM NACL (MG. %)	SERUM K+ (MG. %)	SERUM PROTEIN (GM. %)	REMARKS
March 7, 1937								
13.8	16.05	21.52	64.2	–	–	18.65	–	Burned, left side only, 6:24-6:37 P.M.
15.1	17.95	24.03	63.0	–	734	18.65	7.96	
13.7	19.1	25.60	53.5	–	783	18.65	7.88	Burned, left side only, 9:18-9:22 P.M.
7.0	19.81	26.55	26.9	–	763	22.95	7.55	
12.5	21.65	29.05	43.0	–	724	17.02	7.36	170 c.c. air intravenously at 3:00 A.M. Dead at 3:32 A.M.
March 22, 1937								
								Burned, left side only, 11:09-11:17 A.M.
13.94	14.68	19.65	70.9	45.9	734	14.05	6.32	Harsh systolic murmur developed over apex at 12:10 P.M.
16.12	17.00	22.08	70.7	48.1	734	14.05	6.42	
16.56	18.35	25.91	63.9	45.9	743	14.05	6.40	
13.76	20.40	27.40	50.2	45.8	734	11.24	6.35	
13.65	22.3	29.90	45.7	45.3	754	16.85	6.17	910 mg. pentobarbital at 9:41 P.M. Dead at 9:50 P.M.
April 5, 1937								
								Burned, left side, 11:46-11:50 A.M.
16.3	18.62	24.98	65.2	–	694	15.2	7.22	Burned, right side, 11:56-12:05 P.M.
11.44	18.4	24.65	46.4	–	724	–	7.85	
8.82	21.15	28.2	31.27	–	714	16.1	7.66	
16.34	23.6	31.68	51.5	–	754	15.8	7.04	650 mg. pentobarbital intravenously at 8:55 P.M. Dead at 9:40 P.M.
Splenectomized April 2, 1937								
–	–	–	–	–	641	–	6.31	Burned, left side only, 10:31-10:41 A.M.
8.71	17.45	23.4	37.2	–	–	–	–	
			–	–	–	–	–	
6.24	19.35	24.1	24.1	–	623	–	7.00	
				–	–	–	–	
1.53	19.85	26.55	5.76	–	473	–	7.02	325 mg. pentobarbital 11:07 P.M. Dead at 11:12 P.M.
Splenectomized April 2, 1937								
								Burned, left side, 11:45-11:51 A.M.
7.48	16.20	21.75	34.4	54.4	–	–	–	Burned, right side, 11:51-11:56 A.M.
3.30	17.70	23.65	13.95	54.2	–	–	–	
1.65	23.15	31.05	5.32	50.2	–	–	–	
0.26	24.13	32.35	0.81	46.3	–	–	–	
–	23.70	31.80		–	–	–	–	Dead at 6:57 P.M.

TABLE I—

TIME (HR., MIN.)	WEIGHT (KG.)	RECTAL TEMPERATURE (° F.)	BLOOD VOLUME: PLASMA (C.C.)	% CHANGE	CELL (C.C.)	% CHANGE	TOTAL BLOOD (C.C.)	% CHANGE	HEMATOCRIT (VOL. %)	PULSE (PER MIN.)	SYSTOLIC BLOOD PRESSURE (MM. HG)	RESPIRATIONS (PER MIN.)
Dog 7.—May 19, 1937.												
Initial												
10:15 A.M.	28.94	96.0	1170		1125		2295		50.5	72	132	13
1 00	–	95.2	748	-36.6	962	23.4	1770	-22.9	57.7	150	96	16
2 40	28.44	95.4	715	-38.9	1180	+ 4.9	1895	-17.3	62.3	158	98	14
4 57	28.22	96.3	598	-48.8	1047	- 6.9	1645	-28.3	63.7	168	110	12
9 42	–	101.0	543	-53.5	977	-12.8	1520	-33.7	64.2	222	96	24
12 01	27.44	103.4	543	-53.5	1037	- 7.8	1580	-30.8	65.7	234	54	32
Dog 8.—July 9, 1938.												
Initial										120	140	17
10:05 A.M.	24.18	101.4	1050		1220		2270		55.0			
0 50	–	102.7	–		–		–			142	120	100
2 28	23.40	104.2	875	-16.65	1130	- 7.37	2005	11.65	56.5			
6 46	23.20	106.2	770	-26.7	1060	-13.1	1935	- 1.46	60.3	194	140	70
7 25	–	106.6	–	–	–	–	–	–	–			
8 41	23.20	106.6	–	–	–	–	–	–	–	214	120	120
										244	98	90
										254	70	76

BLOOD GASES

Accompanying the increasing hematocrit values there was a consistent increase in the hemoglobin concentration of the blood in all cases, greater in the nonsplenectomized than in the splenectomized animals. The oxygen content of venous blood fluctuated somewhat in a manner parallel to the changes in the circulating red cell volume. In only two nonsplenectomized animals, 1 and 3, were low values observed. In all of the splenectomized animals there was a marked and progressive decrease in the oxygen content of the venous blood, terminal values of less than three volumes per cent being observed in Experiments 6, 7, and 8.

Due to the increase in oxygen capacity resulting from the rise in hemoglobin concentration and to the decrease in oxygen content, the oxygen saturation fell, terminal values of 43, 45.7, and 51.5 per cent having been observed in nonsplenectomized Animals 1, 2, and 3, in all of which the experiment purposely was terminated. The decrease in oxygen saturation was much more pronounced in the splenectomized than in the nonsplenectomized animals, in that initial levels were lower and the oxygen saturation fell more rapidly and to lower levels. In

CONT'D

OXYGEN (VOL. %)	HEMOGLOBIN (GM. %)	OXYGEN CAPACITY (VOL. %)	OXYGEN SATURATION (%)	CARBON DIOXIDE (VOL. %)	SERUM NACL (MG. %)	SERUM K+ (MG. %)	SERUM PROTEIN (GM. %)	REMARKS
Splenectomized March 24, 1937								
								Left side burned 10:48-10:53 A.M.
11.16	14.78	19.8	56.6	54.0	692	15.0	7.40	Right side burned 10:53-10:57 A.M.
–	–	–	–	–	–	–	–	
16.77	17.70	23.70	70.7	45.7	–	–	–	
9.82	18.40	24.65	39.8	38.5	698	15.0	7.21	
11.67	17.85	23.95	48.8	38.0	706	14.8	6.80	
2.30	17.15	23.0	10.0	52.0	–	–	6.80	Died at 11:04 P.M.
Splenectomized June 21, 1938								
A 13.0	17.48	23.4	55.5	44.5	609	–	6.49	Burned, left side, 10:38-10:44 A.M.
V 11.0	17.48	23.4	48.0	45.5				Burned, right side, 10:44-10:49 A.M.
A 16.2	19.8	26.6	62.3	43.0				
V 15.4	20.4	24.35	56.3	44.5				
A 11.8	18.4	24.65	47.9	46.0	659		6.77	
V 10.9	18.4	24.65	44.2	48.0				
A 8.4	19.35	26.0	32.3	39.0	659		5.88	
V 7.8	19.61	26.3	29.6	40.0				
A 6.4	20.6	27.6	23.1	24.0				
V 5.2	20.4	27.35	19.0	27.4				
A 3.7	21.8	29.2	12.75	50.0				Died at 6:52 P.M.
V 2.9	22.0	29.5	9.83	51.0				

Experiments 6, 7, and 8, which succumbed to the experimental procedure, oxygen saturation was less than 10 per cent just prior to demise. The carbon dioxide content of venous blood changed but little in non-splenectomized animals, the value in the terminal blood sample being about the same as the initial level. In the splenectomized animals there was some lowering of the carbon dioxide content as the experiment progressed, with a terminal rise as respiration failed. A parallel between the lowering of the carbon dioxide content of venous blood and the terminal rise in the respiratory rate was observed.

BLOOD ELECTROLYTES

The concentration of sodium chloride in the blood serum varied little, but, due to the decrease in plasma volume occurring during the experiment, the total amount of this salt present in the circulating blood stream was reduced greatly at the end of the experiment, even in those cases in which the total blood volume was little changed (Experiments 1, 2, and 3). The concentration of serum protein was within normal limits in all experiments, but there was a tendency for the concentration to fall as the experiment progressed, which, when considered in the light of the reduction in plasma volume, indi-

cates a considerable loss of protein from the blood stream. In the experiments in which blood potassium was determined, the concentrations were within normal limits and the variations observed bore no relationship to the duration of the experiment, changes in blood volume, or eventual collapse of the circulation.

PATHOLOGIC FINDINGS

Necropsies were completed within two hours of death. At post mortem in the animals traumatized on one side only paper patterns of the burned areas were obtained for comparison of the amount of surface area involved on the normal and affected sides. In these animals the skin and subcutaneous tissues over the burned area were excised and the weight of this tissue mass compared with the weight of a corresponding area of skin and subcutaneous tissue excised from the normal side as a basis for the estimation of the amount of fluid extravasated into the burned area.

The post-mortem findings in all animals were essentially similar. There was severe dehydration, the tissues, muscles, and serous surfaces all being exceedingly dry. There was practically no fluid in any of the serous cavities, nor was there any "pooling" of blood in vessels or organs in the abdominal area.

On opening the body, the cut surfaces of the traumatized area bulged, revealing a gelatinous, edematous mass beneath the skin, somewhat hemorrhagic and of a greenish tinge, undoubtedly due to staining with the Evans blue. Microscopically there was coagulation necrosis of the skin surface and marked edema and occasional areas of necrosis in the subcutaneous tissues, as evidenced by infiltration by polymorphonuclear leucocytes. The chest was contracted and narrowed, and the diaphragms were high. The lungs were collapsed, lying against the posterior thoracic wall, a light pink in color, very dry and containing little air, microscopically showing only slight engorgement of the capillaries in the alveolar walls. The bronchi contained no mucus. In the lower portions of the lungs posteriorly there were frequent areas of purple color, firm and noncrepitant, from the cut surfaces of which air could be expressed. These sections showed, in addition to engorgement of the alveolar wall, infiltration with polymorphonuclear leucocytes and a marked decrease in the size of the alveolar spaces, the picture being similar to an early bronchial pneumonia of the type seen in childhood. The peribronchial lymph glands were greenish in color, due to staining with the dye.

The pericardium and epicardium were glistening and dry, and a few cubic centimeters of thin yellow serous fluid were found in the pericardial cavity. The heart muscle was firm in some instances and slightly flabby in others, but no involvement of the coronary arteries

was found, and, on section, the cut surface of the ventricle was dry and somewhat granular. Microscopically there were no significant morphologic changes in the heart muscle. The valves were normal in all animals except Experiment 3 in which there was an ulcerated lesion on the posterior cusp of the mitral valve about 9 mm. in diameter with sharply demarcated margins and great thinning of the cusp centrally. This dog developed a loud harsh systolic murmur during the course of the experiment. The aorta appeared normal, as did the esophagus and vena cava, which contained very dark, nonclotted blood.

The abdomen was scaphoid. There was no gross evidence of infection at the site of the insertion of the cannula into the femoral artery. The peritoneal surfaces everywhere were dry and glazed looking, and there was no free fluid in the peritoneal cavity. The omentum was in normal position, waxy in appearance, and the mesenteric vessels were narrowed and poorly filled. In general, there was marked ischemia of the entire gastrointestinal tract, the stomach and bowel being pale and practically empty and contracted, except for the rectal ampulla. The rugae of the stomach were elevated with hyperemic summits. There were no frank ulcers in the stomach. The mucosa of the entire small bowel showed varying degrees of hyperemia, with definite ulcerations in the duodenum and jejunum of two animals. Microscopically the pink ulceration was superficial, but there was an area of necrosis as evidenced by a change in the staining property that extended to the submucosa. The linear folds of the colon were reddened, especially in the lower portions. In some animals the fecal contents of the rectal ampulla were grossly blood stained.

The liver surface was smooth, brown in color, and dry. On section little blood could be expressed from the cut surface, which had a somewhat nutmeg appearance. There was no gross necrosis. Microscopically there were scattered areas of engorgement of the sinusoids around the central vein, with some central necrosis as evidenced by polymorphonuclear leucocytic infiltration. The gall bladder was normal in size, and the bile was a greenish brown in color and seemed normal in amount.

The pancreas was dry, pale, and on section the surface was pale and waxy with scattered red punctate areas. There were no abnormalities microscopically.

In the three animals which were not splenectomized, the spleen was contracted, firm, blue in color with scattered raised purple areas on the surface. On section the pulp seemed very friable, and but little blood could be expressed from the cut surface.

The kidneys were firm, normal in size, blue-green in color. The capsule stripped easily and, on section, the cortex was a deep purple with prominent markings. The pyramids were a pink-purple color,

the pelves normal. Microscopically there was albuminous material within the capsular spaces of the glomeruli, and occasional hyaline and granular casts were seen in the convoluted tubules. Colloid degeneration of the convoluted tubules, the cells of which also contained some hemosiderin, was seen. Ureters were normal. The bladder was empty, contracted, very firm, and there was slight hyperemia of the mucosa but no ulceration, hemorrhages, or calculi.

The adrenals were ivory white, very dry and firm. On section no gross abnormalities of either cortical or medullary portions were evident. In all instances, however, microscopic examination revealed invasion of the cortex by polymorphonuclear leucocytes, involving the zona fasciculata principally and to a lesser extent the zona reticulosa. These lesions are illustrated in Fig. 2. In only one adrenal was there even slight polymorphonuclear invasion of the zona glomerulosa, and this was limited to one small area immediately adjacent to the damaged area in the cortex.

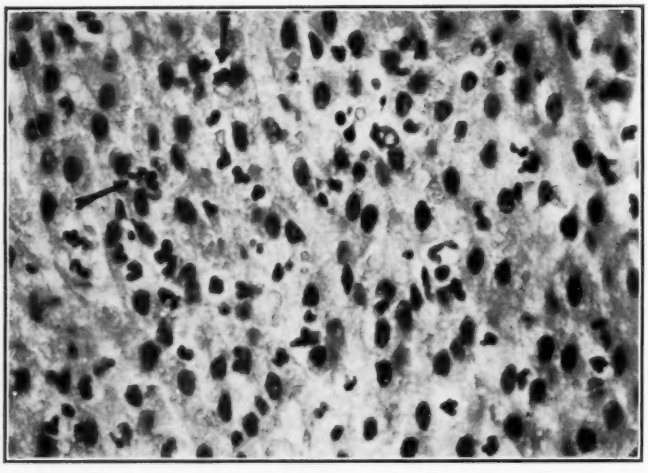


Fig. 2.—Lesions in the adrenal cortex. Section of adrenal from Experiment 7 (×70), hematoxylin and eosin stain, shows the region of the zona fasciculata in which the infiltration with polymorphonuclear cells is most intense. The leucocytes are seen lying between the cortical cells and, in the areas indicated by the arrows, within the cortical cells.

Grossly the membranes of the brain were smooth and glistening, the dura was intact, and there was no evidence externally of any gross hemorrhage. There was little fluid in the subarachnoid spaces. The hypophysis appeared normal and was not stained by the dye.

Histologic studies of the brain of Experiment 8 showed evidence of a diffuse circulatory disturbance. Sections stained by the benzidine technique of Lepehne and Pickworth prepared according to Method 2 in the modification employed by Doherty, Suh, and Alexander[23] showed the entire vascular bed to be congested, the vessels being densely packed with red blood corpuscles, without, however, any marked

dilation. There was some "bead" deformity of the vessels, especially those near hemorrhagic areas. Scattered throughout the brain were small hemorrhages, ranging in severity from perivascular sheath hemorrhages to small perivascular and parenchymatous hemorrhages with extravasation of red cells into the adjacent brain tissue, being most frequently found in the bases of the hemispheres, notably in the prepyriform and entorhinal areas, in the medial and dorsal portion of the thalamus, around the aqueduct of Sylvius, in the corpus callosum, and in the white matter of the cerebellum. These changes are shown in Fig. 3.

In Experiment 2 sections stained by the technique of Masson and Mallory confirmed the findings in Experiment 8, there being dense packing of the vessels with small perivascular and parenchymatous hemorrhages in similar regions with "bead" deformity of some of the vessels.

No cellular damage was seen in any of the brains examined, the cell nuclei everywhere being pale and clear and the cell margins and dendritic processes being sharply defined, nor was there any evidence of glial damage or proliferation.

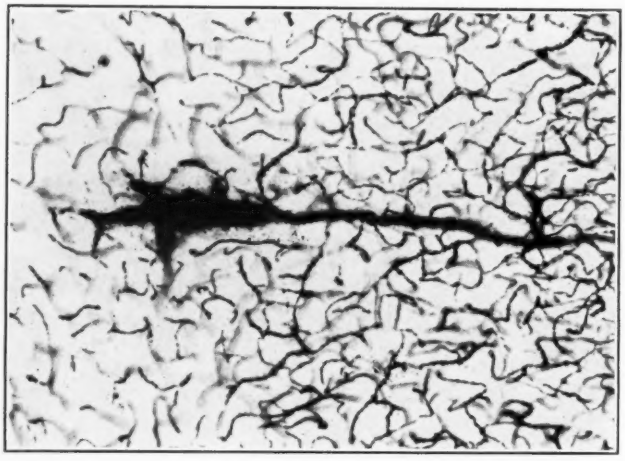
In Experiment 6 evidence of healed previous brain damage, characterized by organization of previous perivascular hemorrhages with thickening of the adventitia and perivascular lymphocytic infiltration, was found. These lesions were obviously old and could have had no relation to the trauma under investigation. In this animal, however, fresh hemorrhages of the type described above were found in the region of the aqueduct of Sylvius.

DISCUSSION

The striking characteristic of the behavior of the circulation in these animals was the maintenance for long periods of arterial blood pressure well above shock levels, even though the animals were severely traumatized. However, demise was due to a complete breakdown of the respiratory and vascular mechanisms, and the extent to which the observed changes contributed to this collapse may be of significance in the consideration of the causes of death in burns.

All of these animals experienced drastic reductions in circulating plasma volume. It may be argued that the presence of a large area with extensive capillary damage might influence the disappearance of the dye from the blood stream and hence the accuracy of the plasma volume determinations. With the exception of values obtained in Experiments 5, 6, and 7 by the short indirect method[16] immediately following the thermal trauma, all of the plasma volume determinations were made with individual dye injections. In a previous communication[17] it was pointed out that the plasma volume, since it is based upon the dye dilution value obtained by the extrapolation of the slope of

A.



B.

C.

Fig. 3.—Lesions in the brain. Sections are of fresh material from Experiment 8, stained by the benzidine technique of Lepehne and Pickworth to show the vascular bed. *A*, Occipital cortex (×24). There is marked congestion. The capillary bed is composed largely of loops rather than straight branching vessels. The vessels are densely packed with red cells but not dilated, averaging 6 μ in diameter. *B*, Area entorhinalis (×70). A perivascular sheath hemorrhage in a severely congested cerebral vascular bed is shown. *C*, Corpus callosum (×60). A small perivascular hemorrhage with extravasation of red cells into the parenchyma of the brain is shown. These lesions are evidence of congestion and stasis in the cerebral vascular bed and hence of some degree of anoxia of brain tissue.

disappearance of the dye from the blood stream to the time of injection, represents the plasma volume existing at the time of injection, and the accuracy of the result is but little influenced by variations in the rate of dye disappearance. Hence the recorded values are reliable.

These reductions in plasma volume resulted from large uncompensated losses of body fluids. In Table II are shown the losses in plasma volume and of tissue fluid into the damaged areas. In this analysis it is assumed that all weight lost may be regarded in terms of body fluid and that all body fluid lost either from the lungs and kidneys or as edema must have passed through the circulating blood stream. Total fluid loss is, therefore, equal to the weight loss plus the weight of the edema fluid, and tissue fluid loss is equal to total fluid loss minus the reduction in plasma volume. Total fluid loss, of which the greater portion was tissue fluid, was 7.24, 7.29, and 6.08 c.c. per hour per kilogram of body weight in Experiments 1, 2, and 5 respectively.

TABLE II

TOTAL FLUID AND TISSUE FLUID LOSSES

EXPERIMENT NO.	WEIGHT (KG.)	WEIGHT LOSS (GM.)	URINE OUTPUT (C.C.)	FLUID LOSS FROM LUNGS (C.C.)	FLUID LOSS INTO DAMAGED AREA (C.C.)	TOTAL FLUID LOSS		REDUCTION IN PLASMA VOLUME (C.C.)	TISSUE FLUID LOSS (C.C.)
						(C.C.)	(C.C. PER HR. PER KG.)		
1	19.65	450	75	375	960	1,410	7.24	284	1,126
2	32.3	1,210	67	1,143	1,160	2,370	7.29	645	1,725
5	22.44	580	63	517	900	1,480	6.08	608	872

It was observed that the rate of disappearance of the dye was more rapid in the early determinations than in those carried out near the end of the experiments, and we regard this as evidence that in the latter part of the experimental period less and less fluid was passing from the blood stream into the traumatized area. This is in keeping with the observation that the greater part of the swelling in the traumatized areas occurred very shortly after thermal trauma. That the plasma volume remained reduced during the latter portion of the experiments, even though the rate of extravasation of fluid into the damaged area was diminished, is evidence of exhaustion of the body fluid reserve. Further evidence of the severity of the dehydration is the almost complete anuria which developed in all these animals, together with the rise in the specific gravity of the urine, even during periods when the systolic blood pressure was well sustained (Table III).

Total blood volume was sustained at very nearly initial levels in the three nonsplenectomized animals, 1, 2, and 3, in spite of the reduction in plasma volume, by a large increase in the volume of circulating red cells. No marked fall in the systolic blood pressure was observed, and it seemed evident that these animals could have survived beyond

TABLE III

SPECIFIC GRAVITY AND CHLORIDE CONTENT OF URINE
EXPERIMENT 8

TIME (HR., MIN.)	AMOUNT (C.C.)	SPECIFIC GRAVITY	CHLORIDE CONTENT (GM. VOL.)
9:30 A.M. July 9, 1938	13	1.045	.0416
20	12	1.047	.0408
45	22	1.050	.0880
1 31	12	1.050	.0024
2 22	13	1.043	.0013
2 50	5	1.066	.0
5 24	4	1.090	.0
8 31	12	1.075	.0

the induced end of the experiment. In these animals the oxygen content of venous blood was well sustained and, while there was a fall in oxygen saturation, it seems evident that the oxygen-carrying mechanism was functioning fairly adequately since respiratory rates did not rise markedly nor did venous carbon dioxide content fall appreciably. In marked contrast to this situation the total blood volume of the splenectomized animals progressively decreased throughout the experiment, having been reduced 33.6, 41.6, and 30.8 per cent from initial levels at the time of death in Experiments 5, 6, and 7 respectively. In the splenectomized animals the percentage reduction in plasma volume was somewhat greater than in the nonsplenectomized animals, but the reduction in red cell volume was chiefly responsible for the marked reduction in total blood volume. In these animals systolic blood pressure, in general, paralleled the course of total blood volume, gradually falling as the experiment was continued until, in Animals 6, 7 and 8, when levels of from 50 to 70 mm. of mercury were reached, there was a rapid and fatal circulatory collapse preceded by a sharp rise in respiratory rate. Just prior to collapse, the total blood volume was reduced 41.6 and 30.8 per cent in Experiments 6 and 7 respectively.

With the increase in hematocrit, there was an increase in the hemoglobin concentration and, therefore, in the oxygen capacity, less marked in the splenectomized than in the nonsplenectomized animals. Venous oxygen content, however, fell progressively to extremely low levels, the oxygen saturation being less than 10 per cent at the time fatal collapse occurred in Experiments 6, 7, and 8. It is evident that the efficiency of the oxygen-carrying mechanism in these animals was seriously impaired. Respiratory rates rose rapidly prior to collapse and venous CO_2 values fell in relation to the increase in respiratory rate.

No marked change in concentration of serum chlorides occurred, but due to large reductions in plasma volume the total amount present in the circulation was greatly lowered. This indicated a large loss of

sodium chloride into the burned area. The concentration of serum protein was diminished terminally in five of the six experiments in which serum protein determinations were done. Here again, due to decrease in plasma volume, the total amount of serum protein was greatly reduced. On the basis of estimation of the amount of fluid (Table II) extravasating into the traumatized area, assuming the protein content of the edema fluid to be approximately equal to that of the blood serum at the time of injury,[24] we compute that the loss of protein into the damaged area was far greater than the reduction in the amount of serum protein in the circulating blood volume. This fact strongly suggests that, in these animals, there existed a source of proteins available for rapid mobilization.

No significant change in concentration of blood potassium occurred, although considerable amounts may have been lost into the damaged areas.

While no determinations of the pH of the blood were made, it is probable that no severe degree of acidosis developed in the nonsplenectomized animals in which respiratory rates were not markedly accelerated, and in which venous CO_2 values and chloride concentrations were normal, but there may have been moderate acidosis terminally in the splenectomized animals.

Several factors must be considered as bringing about the peripheral vascular collapse seen in these animals. Reduction in blood volume with the disparity between volume capacity and circulating blood volume described by Moon[25] is known to produce peripheral collapse. Exactly this picture was described by Gibson and Kopp[26] in patients undergoing artificial fever treatments in whom severe reductions in blood volume preceded profound vascular collapse. It was observed that, as the experiments progressed, there was very little blood in the large peripheral veins, the jugular vein exposed for sampling being partially collapsed. The poor venous return manifested by this lowering of venous pressure could not but aid in the development of cardiovascular failure. It should be said, however, that in all animals in which death occurred respiration was the first to fail, the heart continuing to beat for several minutes after the complete cessation of respiration.

In all the animals which succumbed, a profound anoxia, as evidenced by extraordinarily low oxygen saturations of venous blood, was observed prior to collapse, even though the respiratory rate was markedly elevated at this time. It is hardly possible that the oxygen tension in the tissues, and particularly in the brains, of these animals could have been adequate for proper oxygenation of cells.

In the opinion of Dr. Leo Alexander the perivascular and parenchymatous hemorrhages seen in Experiments 2, 6, and 8 are fresh, and it is reasonable to assume that in point of time they are related

to the duration of the experiment and that they may be considered as evidence of the existence of areas of stasis in a severely congested cerebral vascular bed.

In an effort to evaluate the influence of anesthesia on brain tissue, a control experiment was done using a normal, healthy male shepherd dog weighing 19.1 kg. Anesthesia was induced by the intravenous administration of 32.5 mg. of pentobarbital (nembutal) per kilogram of body weight and was maintained by supplementary doses for a period of ten hours. The animal was then sacrificed by an additional intravenous injection of 975 mg. of pentobarbital, and the brain was removed immediately for study. The sections of the brain of the control animals showed hemorrhages similar to those seen in the brains of the animals subjected to thermal trauma. One significant difference existed. The sections from the control animal showed capillary dilatation in addition to the hemorrhages; whereas, the sections from the traumatized animals demonstrated capillaries of normal size. It is suggested that the degree of dehydration present in the thermally traumatized animals counteracted the vasodilatory effect of pentobarbital. No areas of injury to motor or ganglion cells of the kind described by Gildea and Cobb[27] were observed. It is possible, however, that there may have been some functional damage to nerve cells, since death occurred suddenly, and there may have been insufficient time for the development of morphologic changes.

Although the vascular lesions in the brains of the experimental animals cannot be attributed entirely to the effects of thermal trauma, it is probable that the degree of anoxia, as evidenced by the amount of congestion and the frequency of the lesions, was more severe, and the functional damage to nerve cells was greater in the experimental than in the control animal.

Microscopically the adrenals showed damage to the cortical areas characterized by infiltration with polymorphonuclear leucocytes, to a marked extent in the zona fascicularis and to a less extent in the zona reticularis. The adrenals of a control animal, subjected to nine hours of anesthesia with pentobarbital, showed no abnormalities microscopically. Lesions similar to those seen in the adrenal cortex of these animals have been described by Hartman[28] in animals which died following artificial hyperpyrexia. However, our experiments were acute and sufficient time may not have elapsed for the development of morphologic changes in the adrenal medulla. Blood potassium levels were not particularly elevated beyond levels that might result from hemoconcentration and, what changes did occur, probably were of no significance in the production of vascular collapse. Adrenal damage was very probably not of primary importance but cannot be excluded as playing some role in the development of shock in these animals.

The continuous anesthesia maintained throughout these experiments had the effect of minimizing the part played in burns by reactions to painful stimuli and in our opinion afforded an opportunity to study the changes in the vascular and respiratory systems in the absence of a primary neurogenic factor.

No evidences of infection in the traumatized areas or elsewhere were found at post mortem, and it is, therefore, highly improbable that toxins of bacterial origin played any part in the reaction of the animals.[9] The effect of toxins possibly released from damaged tissue, however, cannot be excluded.[8]

All of the animals had some degree of elevation of body temperature, but in no case was there a rise to fatal levels. The significant effect of the fever probably was an increase in metabolism, as has been shown to occur in typhoid fever by Dubois[29] and in artificially induced fever by Bazett.[30] As a result, the oxygen requirement of these animals must have been increased. With increase in body temperature and oxygen consumption, heat production increases and, since these animals lost heat principally by evaporation of the water content of alveolar air, an increase in the loss of body fluid resulted.

It is evident that no single one of the above factors was solely responsible for the shock which terminated the existence of these animals, but that shock occurred as the result of the final failure of the organism to contend with certain primary reactions to the trauma sustained and secondary disturbances following inevitably in the train thereof. The train of events leading to the establishment of a vicious cycle may be described as follows:

Primary Reactions.—

1. As a result of alterations of the permeability of the capillaries in the traumatized tissues there is an immediate and considerable extravasation of fluid, which may be considered as slightly diluted blood serum containing many red cells, into the damaged area.

2. Since this fluid is withdrawn directly from the blood stream, an immediate and considerable reduction in the circulating plasma volume results, bringing about, in addition to a lowering of the total blood volume, a marked hemoconcentration.

3. A rise in body temperature occurs, due primarily to the thermal input accompanying the application of heat and secondarily to an increase in body heat production, and with this an increase in the oxygen consumption.

Compensatory Reactions.—

4. Extracellular fluid moves into the circulating blood stream in obedience to the laws of electrolytic equilibrium and partially replaces fluid losses from the plasma.

5. As the oxygen requirement increases and the oxygen saturation falls (see No. 7 below), respiratory activity becomes increased, so that the rate of fluid loss from the lungs increases, imposes an additional drain upon the fluid reserve, and brings about further decreases in plasma volume, further hemoconcentration, and a tendency toward an acidosis.

6. The hemoconcentration brought about by reduction in the plasma volume is augmented by a considerable increase in the total volume of the circulating red cells. The increased viscosity of the blood resulting therefrom acts as an impedance to blood movement and impairs gas exchange between tissues and blood. When the available reserve of red cells is exhausted, continued fluid loss brings about a further reduction in total volume.

7. As a result of hemoconcentration, acidosis, and slowed blood flow through the lungs, oxygenation of arterial blood becomes increasingly inefficient, tissue anoxia with a compensatory hyperventilation occurs, and the rate of fluid loss, consequent reduction in plasma volume, and hemoconcentration increase.

In the vicious cycle thus established a point is reached eventually at which the circulating volume is no longer adequate for maintenance of venous return to the right heart, and cardiac function suffers. At the same time tissue anoxia in the brain produces paralysis of respiratory and vasomotor centers with, finally, sudden fatal collapse.

Several conclusions of clinical interest as regards the treatment of severe burns in human beings may be drawn from the above discussion. It is clear that the chief factor in initiating the train of events leading to collapse was the severe and progressive dehydration suffered by these animals. The amount of fluid that can be lost into a severely burned area and the speed with which this loss can take place are striking and emphasize the urgent need for prompt and adequate fluid replacement. The chief effect of an uncompensated loss of fluid into the affected area is a reduction in the plasma volume. It is the consequent hemoconcentration which interferes with the speed of blood flow and with oxygenation that leads inevitably to the vicious cycle of continued fluid loss, hyperventilation, anoxia, acidosis, and eventual respiratory and circulatory failure. Some idea of the amount of fluid required by a severely burned patient can be obtained from a consideration of the total tissue fluid losses of these animals. The loss of tissue fluid was 7.2, 6.56, and 4.57 c.c. per hour per kilogram of body weight in Experiments 1, 2, and 5 respectively, or amounts equal to about one and one-half times the normal plasma volume. Thus, in the case of a man weighing 70 kg. with a severe burn involving about a third of the body surface, based on a tissue fluid loss at the rate of only 5 c.c. per hour per kilogram, the twenty-four-hour fluid requirement would be 8,400 c.c. This conjecture is in keeping with the re-

peated clinical observation that severely burned patients can be given large amounts of fluid parenterally without producing much improvement in blood pressure or clinical state. In addition, it should be remembered that, since the human mechanism for heat dissipation is, to a great extent, dependent upon evaporation of water from the skin, fluid losses in patients with even slight fever may well be in excess of those experienced by these animals. The relatively bloodless and contracted state of the entire intestinal tract seen in these animals would suggest also that water absorption from the stomach and bowel might be impaired in severely burned patients and serve as a good basis for the parenteral administration of fluids, even in large amounts.

The beneficial effect of transfusions in burned patients in all probability is due as much to the additional oxygen carrying material thus supplied as to the replacement of lost serum protein and blood electrolytes. Thus, the animals that could transfuse themselves from their own spleens tolerated the trauma and reactions thereto far better than did the splenectomized animals in which the reserve of available red cells was limited.

A sharp and sudden rise in hemoglobin or red cell count in burned patients should be regarded as an indication for further administration of fluids or for transfusion.

Finally, it should be said that the duration of these experiments, in which the trauma was severe, suggests that the ability of the organism to survive may have been due to the suppression, by the anesthetic, of painful stimuli, permitting the available adjustment mechanisms to function to the fullest possible extent. This suggests the wisdom of the immediate administration of drugs to humans with severe burns to the point of effective and complete relief of all pain. It is suggested that even in patients in shock the inhibition of painful stimuli cannot but have the beneficial effect of permitting bodily adjustments to take place to an extent not possible when the vasomotor and cardiorespiratory systems are reacting strongly to noxious stimuli.

CONCLUSIONS

1. Changes occurring under continuous and adequate anesthesia in the circulating plasma, red cell and total blood volume, in blood oxygen and carbon dioxide content, in the hemodynamics, and in the blood electrolytes were followed in three nonsplenectomized and four splenectomized dogs which were subjected to thermal trauma.

2. The plasma volume was reduced immediately and markedly in all animals, due to a rapid loss of plasma fluid into the traumatized areas. Under further observation this loss continued, and a progressive increase in the insensible water loss brought about a continued reduction in the plasma volume.

3. Large increases in the total number of circulating red cells were effective in maintaining the total blood volume at a level adequate for the support of circulation and for meeting the oxygen requirement in the nonsplenectomized animals. In the splenectomized animals the total number of red cells was reduced and total blood volume was lowered progressively.

4. Arterial blood pressure paralleled the course of the total blood volume and, in three splenectomized animals, fatal circulatory collapse occurred when the systolic pressure had fallen to 50 to 70 mm. Hg, and the total blood volume had decreased about 35 per cent from initial levels.

5. Oxygen saturation of venous blood was reduced in all animals, reaching values of less than 10 per cent prior to peripheral collapse in those animals that succumbed.

6. A progressive uncompensated loss of body fluids and electrolytes may lead to the establishment of a vicious cycle of continued hemoconcentration, congestion, stasis, anoxia, hyperventilation, and acidosis, with eventual fatal respiratory and circulatory collapse.

7. The relation of the reactions observed in these animals to the rationale of the clinical treatment of severely burned humans is discussed.

We wish to express our appreciation to Dr. Darrell Ayer, of the Peter Bent Brigham Hospital, for his assistance in interpretation of the microscopic pathologic findings, and to Dr. Leo Alexander, of the Boston State Hospital, for his examination of the brains and the interpretation of the significance of the cerebral vascular lesions demonstrated by the special staining technique employed.

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PHAGEDENIC ULCER

A Report of Three Cases

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In 1909 Luckett[22] reported an unusual rapidly spreading ulcer of the abdominal wall. The patient, a gardener, noticed a small pimple on the skin of the left lower portion of his abdomen. He picked at this with his fingernail and on the following day observed that a scab covered the site of the former pimple. Removal of the scab revealed a small ulcer which spread rapidly. Six days after he had picked the pimple, the ulcer measured 2 by 3 inches. Twelve days later it measured 5¼ by 10¼ inches and had destroyed the skin covering the greater part of the lower abdomen. As a desperate measure, the entire ulcer, together with the skin well beyond the red area, was excised with the cautery. This treatment resulted in complete and rapid recovery.

Cullen[11] in 1924 described a "progressively enlarging ulcer of the abdominal wall involving the skin and fat, following drainage of an abdominal abscess apparently of appendiceal origin." In the same year Borelli[5] reported a case of "diffuse superficial gangrene of the abdominal wall after laparotomy" and Christopher[9] recorded his experiences with a "severe spreading carbuncular infection of the chest wall following rib resection." The older medical literature contains numerous accounts of enormous spreading ulcers of the skin, some of which are undoubtedly identical with these lesions, as has been pointed out by Bartlett[4] in her excellent review of the subject; but the interest of modern surgeons appears to date from the reports cited above.

Since 1924 descriptions of many closely allied lesions have appeared in the literature.[1-3, 6-8, 10, 12-21, 23-29, 31-36] The majority have occurred after drainage of an intra-abdominal abscess or an empyema. Some, however, have followed drainage of abscesses in other locations; i.e., the axilla, the groin, and the breast. A few are reported as having arisen from small cuts or scratches. Their rarity has made it difficult to study them and most workers have failed to realize that the cases in the literature, which are usually cited as examples of one disease, represent at least two clinical entities. Meleney has described two distinct types: one he calls progressive bacterial synergistic gangrene[25, 26] and the other chronic undermining nongangrenous burrowing ulcer.[27-29]

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The first type, which usually follows the drainage of a peritoneal abscess or an empyema, is essentially a lesion of the skin. It consists of an outer red zone of skin within which is a purple zone. The skin on the central side of the purple zone is gangrenous and gradually liquefies, leaving an ulcer whose base is composed of dirty gray granulation tissue. The process spreads gradually but relentlessly, and unless it is checked by suitable treatment may denude the entire abdomen or back. From this ulcer Meleney has isolated two organisms:[6, 25, 26] a microaerophilic nonhemolytic streptococcus and a *Staphylococcus aureus.* The streptococcus is found in the spreading edge while the staphylococcus is said to be present in the more central gangrenous portions. The streptococcus is described as being "microaerophilic"; i.e., it is not an absolute anaerobe but prefers an environment that is at least partially anaerobic. When first isolated it may not grow aerobically at all, but after several transplants may become capable of aerobic growth. The staphylococcus is an aerobic hemolytic *Staphylococcus aureus.*

Chronic undermining nongangrenous burrowing ulcer is primarily a disease of the subcutaneous fat. The drainage tract of a deep or subcutaneous abscess fails to heal. The skin edges become undermined, roll in, and gradually die. Large areas of skin are undermined and destroyed, leaving an ulcer whose base is composed of pale granulation tissue and at whose margins the skin is often undermined for a distance of several inches. Deep sinuses form about large vessels in the axilla or groin and extend into the pelvis. The process also dissects about bones, such as the ribs and sacrum. From these lesions Meleney has isolated a microaerophilic hemolytic streptococcus.[27, 29] It shows the usual cultural characteristics of beta hemolytic streptococci but prefers an anaerobic environment and usually cannot be isolated unless anaerobic methods of cultivation are employed. However, in long-standing cases Meleney has recovered it on aerobic culture and the organism which he isolated anaerobically will grow aerobically after prolonged artificial cultivation.

Both of these lesions are extremely resistant to all ordinary methods of treatment and continue to spread in spite of many different sorts of local and constitutional therapy. It was soon learned, however, that excision of the skin and subcutaneous tissues down to the fascia well beyond the spreading margin of the ulcer would arrest the progress of the synergistic ulcers and permit healing to occur.

The chronic nongangrenous burrowing type also may be cured by complete excision, but the surgical problem is more difficult. The sinus tracts burrow under ribs, between muscles, and down about large blood vessels in such a manner that their surgical excision is exceedingly difficult and often dangerous. It has been necessary, therefore, to cast about for some other method of treatment. Holman[17]

treated several cases successfully with maggots. Meleney recently has advocated the use of zinc peroxide.[27-30] There are two pitfalls in this method of treatment which must be avoided:

1. All preparations of zinc peroxide are not effective, and one must be sure that he is employing zinc peroxide that is potent. A simple way of determining whether or not zinc peroxide is active has been reported by Meleney.[30]

2. Zinc peroxide cannot be expected to kill the bacteria if it cannot reach them. All sinuses must be widely opened and all overhanging edges of skin, undermined ribs, etc., must be cut away in order to obtain an effective application of the material.

In the past one and one-half years there have been two cases of chronic undermining nongangrenous burrowing ulcer in the wards of the New Haven Hospital. We have also treated a third case which cannot be definitely classified.

CASE 1.—C. K., a white male, 25 years of age, was admitted to the New Haven Hospital on Aug. 12, 1936. On March 16, 1934, while at work in a sand pit, he was struck on the right wrist by a stone. The skin was broken and some swelling appeared locally. One week later the original lesion had healed, but the glands in the right axilla became swollen, fluctuant, and tender.

On March 30, 1934, at the Charlotte Hungerford Hospital in Torrington, Conn., an axillary abscess was incised and drained. Instead of healing, the wound became larger and in spite of all sorts of local treatment an ulcer formed which gradually progressed over the thoracic wall. On Oct. 8, 1934, he was transferred to the Bridgeport Hospital. At this time he said the lesion in the axilla was "big enough to put your fist in." Three days after admission to the Bridgeport Hospital the patient's temperature rose to 106° and the lesion spread rapidly down the medial aspect of the arm as far as the elbow. Many methods of treatment were tried without avail and the lesion gradually spread over the thorax anteriorly and posteriorly. In spite of the extensive lesion the patient felt well and was up and about.

When he was admitted to the New Haven Hospital on Aug. 12, 1936, about two and one-half years after the onset of the disease, a large ulcer covered the entire medial aspect of the right arm, the axilla, and an area on the thorax extending from the level of the clavicle above to the tenth rib below and from the costal chondral junction anteriorly almost to the spine posteriorly (Fig. 1). The base of the ulcer was composed of dirty gray granulation tissue. At the edges in some places the skin was adherent to the base, while in others it was undermined for as much as six inches, the subcutaneous fat having been destroyed. In these areas the progressing border of the undermining process was marked by a raised red area which gradually faded off into normal skin. There were small sinus tracts in the axilla extending between the muscles and about the vessels. Motion at the elbow and wrist was normal, but the arm was fixed at the shoulder. Abduction to 20° could be obtained mostly through motion of the scapula.

During the period between Aug. 13 and Aug. 25, 1936, smears taken from the sinuses and from beneath the undermined skin showed gram-positive staphylococci and gram-negative bacilli. Aerobic and anaerobic blood agar plate cultures from the same areas were overgrown with *B. proteus*. Streptococci were seen in the cooked meat tubes but could not be isolated because of the presence of *B. proteus*. In an effort to eliminate *B. proteus*, sterile saline solution was injected through the skin in the red raised portion of the advancing margin. This was then aspirated and cultured. *Staphylococcus albus* was obtained.

On Aug. 26, 1936, under avertin and nitrous oxide anesthesia, the margins of the entire ulcer were excised with the Bovie electric knife. The excision was carried well beyond the red zone, and a dressing of zinc peroxide* was applied according to the technique recommended by Meleney. Transfusions of citrated blood amounting to 1,800 c.c. were given during the immediate postoperative days.

Fig. 1.—Case 1, upon admission August, 1936. The incision in the skin of the back is in the advancing red margin of the lesion. It was made for bacteriologic purposes. The skin between it and the ulcer is all undermined.

Cultures were made from various portions of the excised tissue upon blood agar plates. These were incubated aerobically and anaerobically. Pieces of tissue were also incubated in tubes of cooked meat medium. From these cultures were isolated:

1. Diphtheroid bacilli
2. *B. proteus*
3. A nonhemolytic streptococcus forming large gray colonies and growing equally well aerobically and anaerobically on blood agar
4. A streptococcus which upon some occasions grew better anaerobically than aerobically; on blood agar plates, anaerobically, it formed a gray hemolytic colony; aerobically it formed a green colony with slight or no hemolysis
5. *B. pyocyaneus*
6. Hemolytic staphylococcus growing best aerobically
7. Unidentified gram-positive rods

Following the débridement, most of the ulcer tended to improve and the granulations became healthy. There was still some tendency to undermine the skin posteriorly near the spine. New sinus tracts formed in the axilla.

On Sept. 9, 1936, additional débridement was done in the axilla, consisting of excision of portions of the pectoralis major and minor muscles. At the same time a portion of the ulcer in the region of the spinous processes of the dorsal vertebrae was excised.

The zinc peroxide dressings were continued. On Sept. 12, 1936, it was noticed that a rib on the anterior chest wall was exposed. During the night of Sept. 23

*The zinc peroxide was kindly furnished by the R and H Chemicals Department of the E. I. Du Pont de Nemours and Co., Inc.

there was a sudden severe hemorrhage from an intercostal artery. The undermined rib was removed, and the patient was given a transfusion of 500 c.c. of citrated blood on Sept. 30. Cultures taken from the tissue excised were overgrown with *B. proteus.*

Azochloramid dressings were substituted at this time as the supply of zinc peroxide had been exhausted. Epithelium began to grow in from the margins posteriorly and inferiorly, but the granulations forming the base of the ulcer did not look healthy enough for grafting. After almost two months without improvement, pinch grafts were applied to several small areas as a test. These promptly disintegrated. On Dec. 4, 1936, there was a sudden rise of temperature and the right arm became swollen and tender. A few days later two sinus tracts which had tunneled down the arm were opened. By Dec. 14 a series of abscesses had formed on both the upper arm and forearm. These were opened and the thin gray pus which was obtained showed streptococci upon smear. Cultures revealed the organisms to be hemolytic streptococci capable of both aerobic and anaerobic growth, but growing more profusely anaerobically.

Fig. 2.—Case 1, June 1, 1937. There is still a small ulcerated area in the axilla.

From this time on the dressings were taken over by one person. They were done meticulously and zinc peroxide was resumed according to the technique recommended by Meleney. Only enough material was available to cover the arm and axilla so the remainder of the lesion was dressed with vaseline gauze. The entire lesion was cleaned daily by a profuse irrigation of physiologic saline solution.

By Dec. 23 the entire lesion was clean and on Jan. 4, 1937, pinch grafts were placed under the granulations over the entire back. Unfortunately zinc peroxide was not available and a vaseline gauze dressing was applied. The patient was given a transfusion of 600 c.c. of citrated blood following the operation. In spite of movement of the chest and the difficulty of preventing the patient from lying on the grafted area, most of the grafts took. Some of the grafts began to regress about Jan. 9, however, and Dr. Meleney, who saw the patient at this time, kindly made arrangements to procure enough zinc peroxide to cover the entire area daily.

The posterior aspect and the arm improved steadily. But on Feb. 2 an area of redness appeared over the sternum, and it was found necessary to excise the fifth, sixth, and seventh costal cartilages which had been undermined and honeycombed. The granulations did not seem to grow in any portion of the wound though the grafts grew slowly. On Feb. 15 the anterior surface of the ulcer was covered with small pinch grafts which were placed under the granulation tissue and the patient was given 500 c.c. of citrated blood. These grafts took fairly well. On Feb. 25 grafts were placed on the back where previous grafts had been lost. By this time the greater portion of the lesion was epithelialized, although there were a few nongranulating areas near the sternum where grafts would not take. On April 19, 1937, it became necessary to excise a small sinus tract together with a portion of a costal cartilage.

Fig. 3.

Fig. 4.

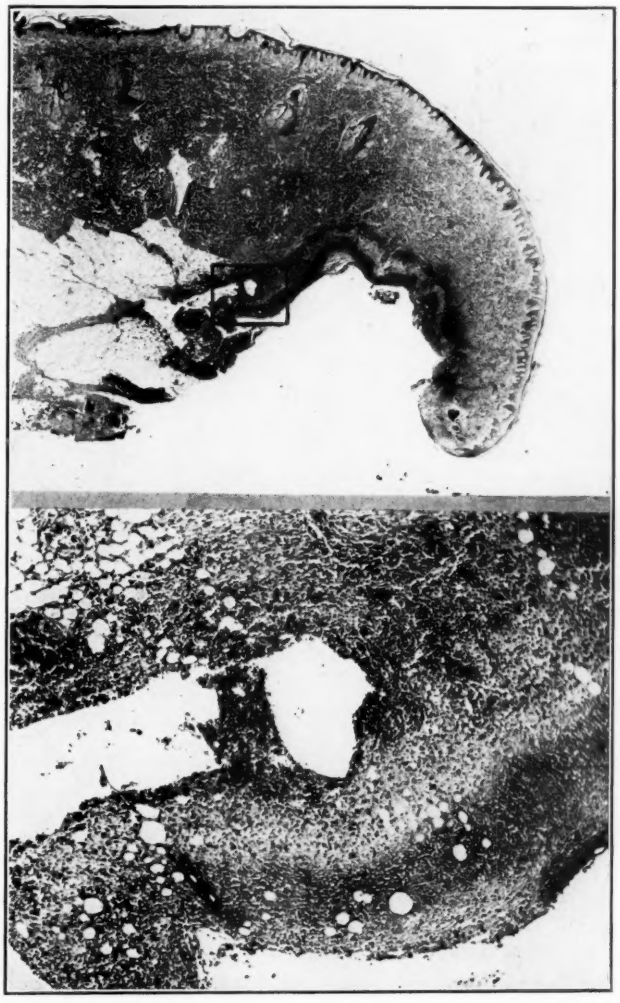

Fig. 3.—Case 1 (hematoxylin and eosin, ×10). Section through margin of ulcer showing destruction of subcutaneous fat with relatively normal epithelium and dermis above.

Fig. 4.—Case 1 (hematoxylin and eosin, ×85). Higher power taken through area indicated in Fig. 3 showing ulcer surface.

By June 1 (Fig. 2) the entire ulcer was healed except for a few small areas 1 to 2 cm. in diameter in the axilla. There seemed to be nothing to excise, and these were treated conservatively. Sometimes a small ulcer would appear in the

center of the old scar, only to disappear quickly. This state of affairs continued until Aug. 8, 1937, when it was decided to excise the area in the axilla. A long sinus tract was found extending high up into the axilla. This was laid wide open, but it healed very sluggishly, and it was not until Oct. 18, 1937, that the lesion was healed and the patient was discharged.

Pathology.—(Figs. 3 and 4.) Sections taken through the margins of the ulcer show that the lesion involves chiefly the subcutaneous fat. The epithelium and dermis are intact and free from cellular infiltration except at the periphery of the overhanging edge. The subcutaneous fat is absent beneath this overhanging edge of dermis and epithelium but as the lesion is followed toward the advancing margin this fatty layer gradually reappears. At first it is necrotic and densely infiltrated in

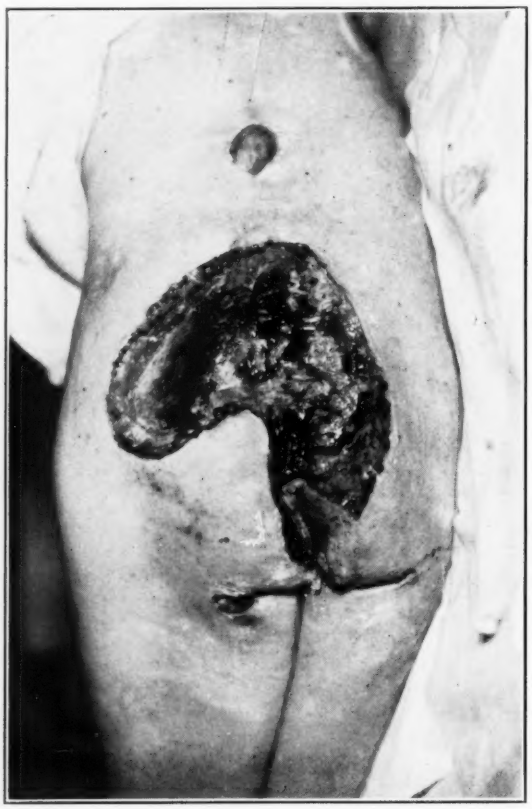

Fig. 5.—Case 2. The ulcer over the sacrum and perineum after débridement Sept. 16.

some zones with polymorphonuclear lymphocytes. As the border of the lesion is approached, the necrosis is absent, leaving merely a wall of inflammatory cells demarcating the limits of the process. The walls of the ulcer are lined by a layer of amorphous necrotic material which is heavily infiltrated with polymorphonuclear leucocytes. Beneath this is a zone of fresh granulation tissue composed of young fibroblasts and budding capillaries. Beyond this zone older granulation

tissue is seen. This is heavily infiltrated with lymphocytes, plasma cells, and rare polymorphonuclear leucocytes. The blood vessels throughout are intact and free from thrombi.

Fig. 6.—Case 2. The area shown in Fig. 5 after healing had occurred.

Case 2.—E. M., a white male, 28 years of age, came to the New Haven Hospital Dispensary on Nov. 22, 1933, complaining of a sore in the right axilla. He stated that six weeks previously a red, tender lump had appeared in the axilla. The patient had applied hot compresses to the lesion, but, when this treatment failed, he squeezed the lump, forcing out a small amount of green pus. The lesion, failing to heal, continued to discharge purulent material. Examination revealed a sinus tract in the right axilla which extended beneath the skin for 3 cm. The entrance of the tract was surrounded by red indurated skin. The only other significant finding upon physical examination was a typical pilonidal sinus. The patient refused treatment that was offered and was not seen again until Oct. 16, 1935, when the skin of both axillae was thick, red, and perforated by the orifices of numerous sinus tracts. These could be traced for only short distances beneath the skin. The pilonidal sinus had become infected and it was possible to express pus from the many small orifices overlying the sacrum. Kahn and Frei tests were negative. An intracutaneous tuberculin test was positive. No cultures were reported. The patient again left the Dispensary and was not seen further until June 11, 1937, when he was admitted to the hospital. During the year and a half he had treated the axillary lesions with hot compresses and had succeeded in healing the one in the right axilla completely. The left axilla, however, was occupied by a larger ulcer whose base was composed of dirty gray granulation tissue. About the ulcer the skin was red and indurated but not undermined, and there seemed to be evidence of healing. The lesion, which

had started in the pilonidal sinus, had spread so that the perianal, lumbosacral, gluteal, perineal, and inguinal regions were occupied by a large ulcer, whose base was composed of dirty gray granulation tissue. The skin edges were undermined several inches in some areas, while in other places there were bridges of skin crossing the ulcer. Sinus tracts led down about the rectum, the sacrum, and dipped between the gluteal muscles.

Fig. 7.

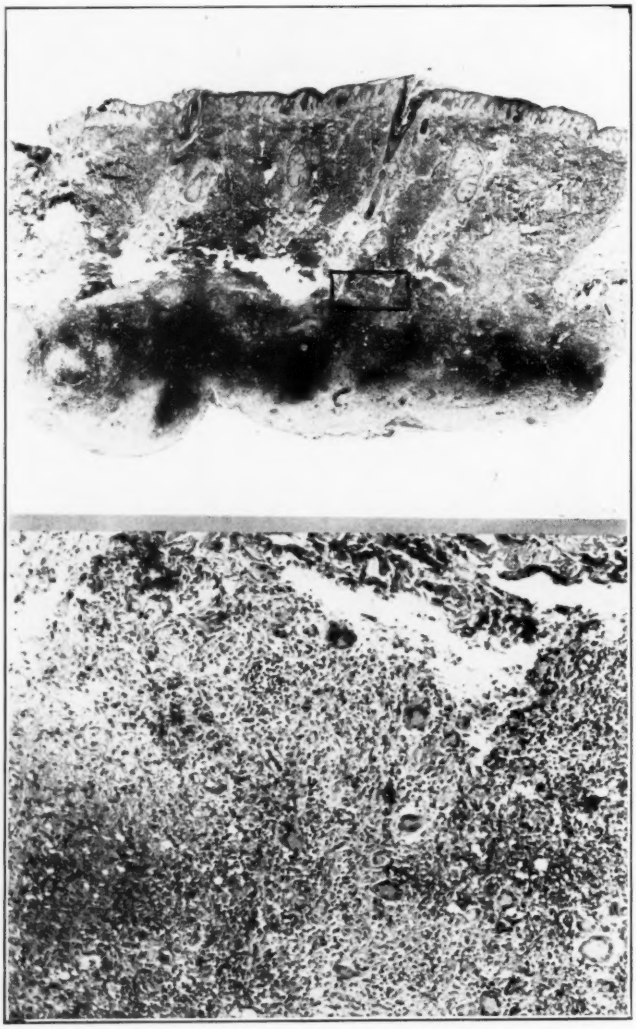

Fig. 8.

Fig. 7.—Case 2 (hematoxylin and eosin, ×10). Section through undermined portion of ulcer margin showing involvement of the subcutaneous fat with relatively normal epithelium and dermis above.

Fig. 8.—Case 2 (hematoxylin and eosin ×125.) High power taken approximately through area indicated in Fig. 5 showing foreign body giant cells.

Aerobic and anaerobic cultures made on June 21 from the lesion in the left axilla and the ulcer over the sacrum were overgrown with *B. proteus*. On June 22 more cultures were taken from many different locations in an effort to obtain some that might be free of *B. proteus*. From these cultures were isolated:

1. Nonhemolytic anaerobic streptococcus
2. *Staphylococcus aureus*

3. Diphtheroid bacilli
4. *B. proteus*
5. Unidentified gram-positive bacilli (aerobic)
6. *Staphylococcus albus*

It did not seem that much etiologic significance could be attributed to such a heterogeneous collection of organisms. On June 29 a piece of tissue was excised from the margin of the ulcer. From the tissue nearest the ulcer were isolated diphtheroid bacilli, *Staphylococcus albus*, and anaerobic nonhemolytic streptococci, while from the tissue near the normal skin were isolated diphtheroid bacilli and *Staphylococcus albus*.

Treatment consisted of tub baths twice daily to keep the lesion clean. Injections of tartar emetic were given intravenously. There was very little change in the lesion over a period of two and one-half months, and on August 25 zinc peroxide dressings, according to the technique outlined by Meleney,[27] were started. After two weeks of this regime, very little improvement was noticed. Therefore it was decided to excise the lesions radically. On Sept. 16 a wide excision of the ulcer in the lumbosacral area was done. The periosteum of the sacrum was exposed and a portion of the rectum lay almost free in the wound (Fig. 5). Zinc peroxide dressings were continued. Granulation tissue appeared in ten days.

On Oct. 19, 1937, the granulating area was covered with pinch grafts, nearly all of which took. By Nov. 29 the entire area was epithelialized. The left axilla had healed meanwhile, with no treatment other than the zinc peroxide dressing. Cultures taken Oct. 23 were overgrown with *B. proteus*.

The ulcer remaining in the perineal and inguinal region was removed on Dec. 2, following which the patient recovered completely (Fig. 6).

Pathology.—(Figs. 7 and 8.) Sections taken through the margins of the ulcer present an appearance identical with that of Case 1 except that the walls of the ulcer are lined by a thick layer of granulation tissue throughout which are scattered many multinucleated giant cells of the foreign body type. These are large cells with pale basophilic cytoplasm containing opaque bodies and with six to fifteen nuclei arranged in a ring about the periphery of the cell.

CASE 3.—L. G., a white male, 75 years of age, was first admitted to the New Haven Hospital on April 27, 1936. He had been well except for abdominal cramps and mild attacks of "indigestion" until ten months before admission to the hospital, when, following a severe attack of "indigestion," he began to lose weight and strength and to have a fever. Four months later a tender mass formed in the right flank and ruptured spontaneously, liberating a quart of pus. The sinus leading to the abscess cavity failed to close. A few months later he began to cough.

Upon admission the patient appeared chronically ill. There was a sinus tract in the right flank which could not be probed beyond 2 cm. but which was evidently longer. A large accumulation of fluid was noted within the right pleural cavity. When this was aspirated, thick green pus was obtained. A smear of the pus revealed a few gram-positive cocci in short chains. Hemolytic streptococcus, *Streptococcus viridans*, and *Staphylococcus albus* were recovered by aerobic culture. No anaerobic cultures were made.

A long series of operations ensued which included catheter drainage of the empyema, rib resection, and partial thoracoplasty in many stages with excision of portions of the parietal pleura in order to close the chronic empyema cavity. The sinus in the right flank failed to close and was explored. It ran down along the lateral abdominal wall to a large mass in the region of the cecum and was found to

connect with the appendiceal lumen. Due to the extent of the mass, it was necessary to resect the terminal ileum, cecum, ascending colon and to anastomose the ileum to the transverse colon. The cecum and appendix were occupied by numerous large papillomas which almost completely occluded their lumina.

The point of special interest here was the development of a spreading ulcer on the chest wall following one stage of the thoracoplasty.

On Nov. 26, 1936, about one month after operation and one week after discharge from the hospital, it was noted that one of the wound margins was being rapidly undermined. An ulcer 3 cm. in diameter formed during the next twenty-four hours, and the patient was admitted to the hospital. Aerobic and anaerobic cultures of pieces of tissue excised from the margins of the ulcer revealed hemolytic *Staphylo-*

Fig. 9.—Case 3, May 30, 1937, just before the final débridement. Note the empyema cavity above the ulcer and the scars of the previous operations

coccus aureus and *Staphylococcus albus*. The margins of the wound were held open with adhesive tape and gauze saturated with H_2O_2 was packed beneath them and changed hourly. The infection was readily controlled by these methods and on Dec. 14 the patient was discharged.

On April 22 a partial thoracoplasty was done and on May 10, 1937, he was discharged to be followed as an out-patient. He was readmitted because of the undermining of the margins at the lower end of the wound on May 24. Aerobic and anaerobic cultures of the ulcer margin at this time yielded diphtheroid bacilli and nonhemolytic streptococci growing equally well aerobically and anaerobically. It was hoped that this ulceration could be controlled as it had been previously by

merely excising the overhanging edges to get exposure and then packing the wound with gauze soaked in H_2O_2. In spite of this treatment, the ulcer spread. On May 28 under general anesthesia a wide resection of the ulcer was done with the Bovie cautery and continuous irrigations of hydrogen peroxide were started. Cultures of the excised tissue yielded the same organism plus *Staphylococcus albus.*

At this time the patient presented a residual empyema cavity with an ulcer 10 cm. in diameter at its inferior margin (Fig. 9). Its base was formed by fascia and its margins by the skin and subcutaneous fat. Laterally the margins were healthy but medially and inferiorly they were slightly undermined and the surrounding skin was raised and red for a distance of 2 cm. beyond the border of the ulcer. Zinc peroxide was applied according to Meleney's technique, but the undermining continued at such a rapid rate that one could note a change in three to four hours. Accordingly, under general anesthesia a really wide excision of the ulcer was done on May 30 and zinc peroxide was applied. Aerobic and anaerobic cultures of the excised tissue yielded the same organism as before plus *B. pyocyaneus* and *B. coli,* which were thought to be contaminating organisms acquired in the hospital.

Fig. 10.—Case 3. The ulcer is completely healed, but there is still a small empyema cavity.

The ulcer improved rapidly after the excision except in one area where the end of a rib was exposed. On June 22 pinch grafts were applied. The grafts took well and epithelium grew in rapidly from the margins.

By July 7 the ulcer was entirely healed except for a small sinus beneath the exposed rib. The rib was resected and thereafter the lesion healed completely. (Fig. 10.)

Pathology.—(Figs. 11 and 12.) Sections through the margin of the ulcer show a necrotizing process involving the epithelium, dermis, and subcutaneous fatty tissue. The edges are lined with necrotic tissue which is densely infiltrated with lymphocytes, plasma cells, and polymorphonuclear leucocytes. In zones where the ulcer has penetrated deeply into the subcutaneous tissues there is a marked tendency for

Fig. 11.

Fig. 12.

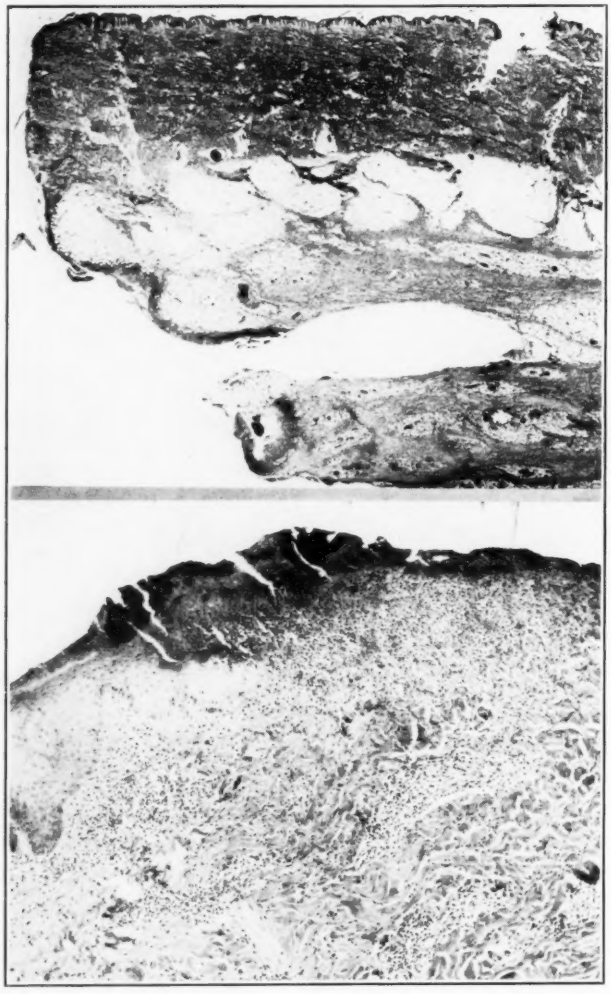

Fig. 11.—Case 3 (hematoxylin and eosin ×14). Section taken through margin of ulcer. Tissue removed at last excision. The sharp vertical edge of epithelium and dermis is due to previous débridement. Note undermining in the subcutaneous fat.

Fig. 12.—Case 3 (hematoxylin and eosin ×85). High power taken through one of the areas of skin necrosis. Note necrotic skin and cellular infiltration of dermis.

the cellular reaction to extend laterally, especially in the fatty layer. In the less penetrating portions the process appears to be confined chiefly to the subepithelial layers. The transition between normal and involved tissues is a gradual one with no wall of inflammatory cells demarcating the advancing margin.

The gross appearance and clinical courses of Cases 1 and 2 were quite similar. These cases, we believe, fall into the group of chronic undermining nongangrenous burrowing ulcers as described by Meleney.[27] The third case was entirely different clinically and seemed to resemble more closely the ulcers Meleney described under the title of progressive bacterial synergistic gangrene.[25, 26] Microscopically, however, except for a marked cellular reaction in the dermis just beneath the epithelium and occasional zones near the edge of the ulcer where the epithelium is necrotic, the picture is not unlike that seen in the first two cases. This ulcer was treated so early and its margins were excised so frequently that it did not have time to develop the typical clinical appearance of either type. For these reasons we are not willing to classify it definitely.

A review of the literature and our own limited experience has convinced us that Meleney's two clinical types of spreading ulcers and perhaps others exist. Whether these are bacteriologic entities in that they are each caused by specific organisms or whether they merely represent the reaction of certain patients to a nonspecific chronic infection, we are not prepared to say. Meleney, as pointed out above, has isolated organisms which he believes are the specific causative bacteria of each type of ulcer.

In the first two cases, which we regard as examples of the chronic undermining nongangrenous burrowing ulcer, the ulcers harbored *B. proteus*. This made all attempts to recover other organisms very difficult and may account for our failure to recover the organisms described by Meleney. In Case 1 streptococci were isolated in pure culture from an abscess when the lesion spread down the patient's arm and upon one occasion from tissue removed during a débridement. These resembled somewhat the microaerophilic hemolytic streptococcus described by Meleney. However, they were recovered from the ulcer proper upon only one occasion and certainly were not present in abundance. Furthermore they grew quite well aerobically.

In the third case we were not bothered with the proteus bacillus and in spite of repeated attempts we were unable to isolate a microaerophilic nonhemolytic streptococcus, or a microaerophilic hemolytic streptococcus.

The cultures described above were made by streaking 5 per cent rabbit's blood agar plates with material from the ulcer. Some plates were incubated aerobically and others anaerobically in a McIntosh-Fildes hydrogen jar. Portions of the tissue removed at operation were also incubated in deep tubes of cooked meat medium from which aerobic and anaerobic plates were made.

In an effort to establish the etiologic significance of the organisms that had been isolated, we attempted to reproduce the lesions in

animals. Numerous guinea pigs were injected subcutaneously with pure cultures and with various combinations of the organisms isolated from the ulcers.

In some cases we succeeded in producing necrosis of the skin which sloughed away leaving an ulcer with undermined edges, but these ulcers always healed promptly and never seemed to have more than a vague resemblance to the progressive spreading lesions in the patients. Furthermore we were able to produce similar ulcers in guinea pigs with mixed cultures obtained from patients whose wounds bore no resemblance to these spreading ulcers.

Some of the combinations of bacteria which produced ulcers in guinea pigs were injected subcutaneously into mice, rabbits, and a dog. The mice died promptly, but in the rabbits and in the dog abscesses formed at the site of injection, ruptured spontaneously, and healed. Pieces of tissue from the spreading margins of the ulcers of the patients were placed beneath the skin of guinea pigs. All of the pigs developed an indurated swelling at the site of the tissue which disappeared after a week or two without ever breaking down.

Holman[17] and Brunsting and others[7] report similar results with their animal experiments, in that they were able with some combinations of organisms to produce lesions in animals which, however, healed promptly. Holman injected into dogs a hemolytic streptococcus and a *Staphylococcus aureus* isolated from a case of chronic nongangrenous burrowing ulcer. He was able to produce a slough at the site of injection, but healing occurred rapidly, and the lesion did not resemble that seen in the patient. Meleney has not reported on any experimental inoculations with the microaerophilic hemolytic streptococcus isolated from the chronic undermining nongangrenous burrowing ulcers but Brewer and Meleney[6] reported the results of some animal inoculations with the organisms isolated from the progressive synergistic type of ulcer. They found that neither the *Staphylococcus aureus* nor the microaerophilic nonhemolytic streptococcus when injected subcutaneously in dogs, rabbits, and guinea pigs would produce a lesion but if they were injected simultaneously a necrotic area resulted which broke down and left an ulcer. In the guinea pigs and rabbits "the process generally reached its peak in three to four days and thereafter subsided. It did not spread progressively as in the human case." In the dog "on the fifth day frank gangrene developed at the injection site of the mixture and on the sixth day it sloughed out, leaving an undermined gangrenous margin. This spread slightly for a day or two and then subsided but showed very little tendency to heal."

SUMMARY

The three cases confirm the general experience of others in regard to the treatment of these ulcers. Ordinary methods of treatment are

usually of no avail and although these lesions, while under treatment, may tend to regress in one area while progressing in another or even regress and remain dormant for a time, they eventually will progress until they destroy the patient unless something radical is done. Before Meleney introduced the zinc peroxide treatment, there were only three or four cases reported in the literature that had recovered without radical excision of the ulcers.

In our three cases zinc peroxide alone was powerless to control the lesion, but, when used in conjunction with surgery, it seemed to be of definite benefit. It was never able to stop undermining of skin, ribs, or muscles alone but once these infected areas were completely removed, the material appeared to prevent recurrence. We have no proof of its mode of action and are unable to decide whether it acts specifically against anaerobic organisms by liberating oxygen, as Meleney believes,[30] or whether it merely provides a comfortable nonadherent dressing which promotes drainage and requires such meticulous care of the wound that healing results.

I wish to express my appreciation to Dr. Stanhope Bayne-Jones for his helpful suggestions, interest, and encouragement throughout the course of the work.

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HYPOSPADIAS

A Discussion of the Subject from the Viewpoint of Reconstructive Surgery and a Report of the Use of a Depilated Scrotal Flap*

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HYPOSPADIAS is a deformity of the male genitals, due to a failure of the embryonal completion of an essential structure, the urethra. The defect is always in the midline of the ventrum of the penis. One case varies from another only in the degree of developmental failure. The degree of the defect varies from a marking on the glans penis to a point where the sex of the individual may be questioned. It is important to note that both penile and scrotal elements may be involved.

In the normal case the function of the urethra is that of a channel of evacuation of the urinary bladder and after puberty it plays an important role in the sex life of the individual.

The function of the scrotum is that of a covering of the testes. It is composed of epithelial structures which contain hair follicles which in this location do not produce until after puberty.

In the site and position of the absent structure appear firm, hard tissue, vestigial bands of the urethra, which inhibit complete erection of the penis and cause a condition called encurvature.

Master surgeons have devoted their efforts to finding ways and means for the reconstruction of a part of the body which has been destroyed or rearranged by the invasion of disease, the application of force, or which has failed to develop congenitally or to grow normally. A splendid literature, discussed under the terms of reconstructive or plastic surgery, has developed and is accessible to any surgeon. Certain principles of procedure and requirements of success in the mobilization of tissue to repair a defect have been developed and generally accepted. The fundamental problem of repair of the deformity of hypospadias is to find tissue from somewhere to supplant the missing structures.

Thus I have analyzed the many operations suggested for the repair of hypospadias, studied the operative steps involved, tried to relate them to these principles and to sift out those steps which do not fit and concentrate upon those that do, in an effort to find the most reasonable solution of the question of method of reconstruction of the deformity.

1. *The Use of the Free Graft.*—The free graft, whatever its form, thin or thick, is severed from its original source of nourishment and transplanted to another part of the body. It is, therefore, potentially dead

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tissue and ideal situations must obtain for the development of new avenues of nourishment, however indirect, or complete or partial loss of the graft follows.

The successful transplant of a free graft depends upon: the use of one kind of tissue, a properly prepared base for its application, and fixation of the graft to the base by some agent of pressure.

In the repair of hypospadias the thin skin graft only has been used and thus the first requirement is readily met.

The preparation of the base of application in hypospadias is made by a trocar, around which the graft is sewn in reverse, being plunged into the body of the penis. The graft is thereby buried in a cleanly and newly prepared base, which meets the second requirement.

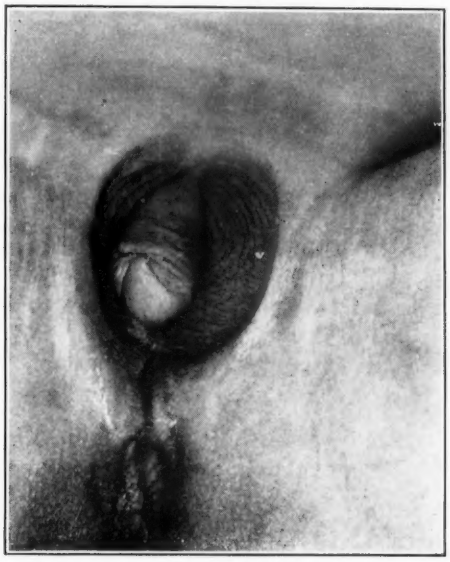
Fig. 1.—At the age of 7 years and 4 months, this boy was admitted to the hospital, in September, 1927, for the repair of hypospadias.

Pressure to produce fixation of the graft is exerted by the inflexible tube of the trocar placed within the soft movable tissues of the organ and its skin covering.

Experience has shown that success in free transplants is best assured where the graft is placed on structures which offer resistance to pressure. The tissues of the genitals are soft, pliable, movable and non-resistant and the possibility of exerting even firm pressure on the graft seems to me to be rather remote.

The use of the free graft in the repair of hypospadias was given a long trial at the beginning of the century by Nové-Josserand. Cecil

comments upon the persistence with which this plan has been followed in spite of inconclusive and varying results. The often demonstrated tendency of a free graft to contract when placed on soft movable tissue



A.

B.

Fig. 2.—*A*, The close-up of the genitals. *B*, The opening is located at the scrotal-perineal junction. By the slightest depression of the tissues the catheter went straight in behind the pubis. First operation for the release of the penis was done, and the patient was discharged in a month.

requires the most meticulous care in aftertreatment, to such a degree that the follow-up attention is as important, if not more so, than the actual performance of the operation. The tendency of the free graft to contract when placed on movable tissues renders its use in the immature patient with hypospadias questionable, because of the possibility, even probability, of encurvature of the penis increasing with future growth of the organ. In other words, the organ grows but the graft does not.

Recently McIndoe has responsored the plan, giving details of operative procedure and aftercare. His argument, however, seems to indicate that he is referring to the adult case of hypospadias, whether the operation be a primary procedure or one secondary to a partial or com-

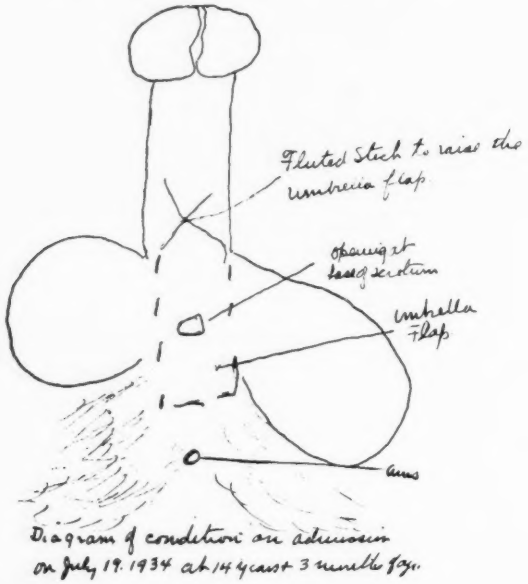


Fig. 3.—He was readmitted in July, 1934, a lapse of seven years since discharge. The penis was satisfactorily released, the opening relaxed also, requiring a channel to be built, the whole length of an approximately mature organ. Every plan of repair incurred a great risk of failure except the stepup operation of Gatewood. A pucker stitch was placed in the umbrella flap and the patient was discharged in eighteen days.

plete failure of a previous operation. The operation can be done but I believe should be attempted only with a full knowledge of the exacting precision required in the preparation of the graft, the possible technical difficulties of insertion and fixation, and the need for ultrameticulous postoperative care.

2. *The Advancement of the Urethra.*—The rationale of the operation of advancement of the urethra is based on the fact that the urethra normally lengthens and shortens with the engorgement and recession of the cavernous bodies of the penis. In advancement of the urethra, the congenitally short urethra is placed in a forward position in the belief that it then will function normally in the new position. How-

Fig. 4.—He was readmitted in November, 1935, at the age of 15 years and 7 months. A photograph was taken on admission. The opening had been advanced to the penile-scrotal junction, was patulous, and the channel was filled by a tangled mat of black hair. The tissues were so elastic and pliable that it was possible to evert parts of the channel at a time. He was sent to the operating room without preparation and a tedious effort made to destroy the follicles with a fine fulgurating needle.

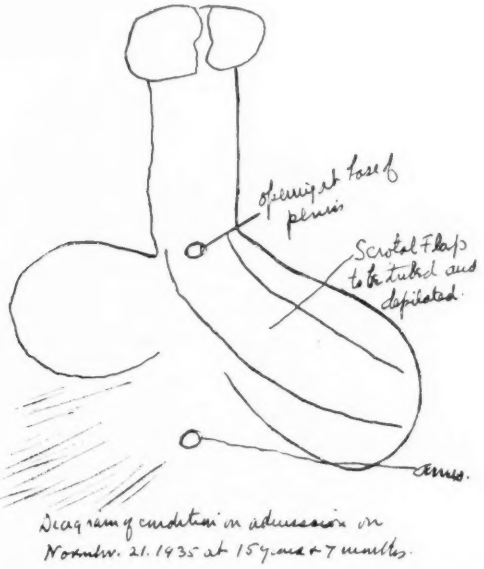


Diagram of condition on admission on
November 21. 1935 at 15 years + 7 months.

Fig. 5.—At the same operation, a flap was plotted on the scrotum, was positively tubed, and the hairs on it treated.

ever, in some cases it is difficult to determine the real length of the malformed urethra. The position of the opening is no index, because this opening itself shows many forms, even to a degree of almost complete stricture.

A.

B.

Fig. 6.—*A*, The picture of the form of the flap. Attention is called to the width of it. *B*, The picture of the puncture wounds. All healed without attention but objections to the method are discussed in the text.

In three weeks the flap was elongated one and one-quarter inches and further attempts to depilate were made, all under local anesthesia.

After school, in June, 1936, he was given another course of depilation and the distal end of the flap was incised, as a first step in its release.

I have positive evidence that the length of the urethra is also a cause for encurvature as well as the presence of the contractural bands distal to the opening. I have felt a deep responsibility in sending a boy out of the operating room with a more severe degree of hypospadias than that with which he entered it. I believe that the first operation for release of the penis is as important, if it is not the most important of all, as any of the operative steps, because the purpose of the repair is not only to build an intact urinary channel but also to construct a competent sexual organ.

A. *B.*

Fig. 7.—In September, 1936, the flap was released, swung up, and attached to the right side of the penis by two series suture lines of removable stitches. *A*, Outside view of the flap; *B*, inside view of the flap; note a well-developed organ.

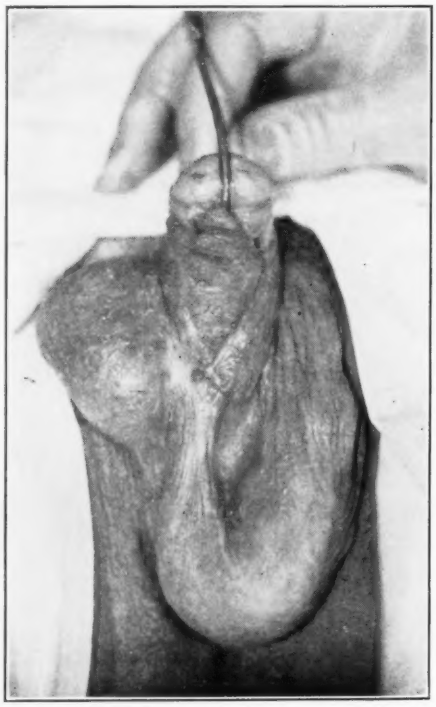
In the operation of mobilization the urethra is made into a single ended pedicle flap and the maintenance of blood supply in the flap must be fully considered. The degree of the deformity, therefore, must influence one in the selection of this plan, a plan necessarily limited to use in the lesser degrees of anterior penile and balanitic hypospadias, cases which I believe are questionable subjects for operation. I have been unable to make myself believe that a congenitally short urethra can be made to stretch the required distance without risk of causing an encurvature of the penis.

3. *The Transplant of Structures.*—If the principles of procedure and requirements for success in the use of flaps (*see* No. 5) and free graft transplants (*see* No. 1) are accepted, it must be concluded that success in the use of the appendix, ureter, vein, bladder mucosa, etc., for a new urethra results not from a technical victory but from a dispensation of providence.

4. *Jump Flap From Outside the Genitals.*—This plan has never received attention.

When the amount of scrotal tissue is considered, why should it be necessary to seek tissue from outside the genitals?

Because of so many questions of selection, possible technical difficulties, or, as in No. 3, when studied from the angle of tissue transplants, impossible procedures, I suggest that Nos. 1 through 4 be put aside and concentration of effort be made on No. 5.



A.

Fig. 8.—Following the transplant of the flap to the penis, he returned to school. In July, 1937, an incision was made on the left side of the penis, the flap split and fixed by two series suture lines, the inner line with hardened catgut, the other with removable stitches. The urine was diverted by a catheter which remained in position for ten days. There is an incompletely descended testicle on the right side. The flap was taken from the left scrotum. *A*, Side view; *B*, front view.

5. *Mobilization of the Tissues of the Genitals.*—The proposed operations include flaps cut from the prepuce, the epithelial envelope of the penis, and the scrotum.

The flap operations have been known, have been discussed and rediscussed for many years by their sponsors. When these operations are broken down into separate technical steps, they are immediately resolved into different combinations of the use of several well-known forms of flap.

The forms may be tabulated as follows:

1. *The Surgically Tubed Flap Positively Sutured.*—The epithelium is on the outside of the flap. Example: The form shown in my illustrated case.

2. *The Surgically Tubed Flap Sutured in Reverse.*—The epithelium is on the inside of the tube.

A. Cut from the ventrum of the penis. Example: The inner tube flap of the Duplay-Thiersch operation, used by Blair-Brown, Cabot, etc.

B. Cut from the dorsum of the penis. Example: The Mayo operation.

C. Cut from the scrotum. Example: The Lowsley-Begg operation.

3. *Sliding Flap.*—Example:

A. The outer flaps of the Duplay-Thiersch operation modified by Cecil into an overfolding flap, which, I believe, is based on the same surgical principle.

Fig. 8*B*.—See opposite page for legend.

4. *Pedicle Flaps.*—

Single Ended:

A. Cut from prepuce. Example: Edmunds, modified by Denis Browne into a recession flap. This flap was used in the 1870's by Van Horn.

B. Cut from scrotum. Example: Immediate transplant, Blair-Browne and Cabot. Delayed transplant, Bucknall.

C. Cut from penis and possibly scrotum. Example: Ombredonne, popularized by Bevan and Gatewood as an umbrella flap, both of which are advanced flaps.

Double ended:

Example: Ombredonne cut from prepuce.

There are not so many forms but when cut from different areas of the genitals it is easy to look upon them as different procedures.

All flaps are acceptable and are wonderful contributions. It is still a marvel to me that we may attempt such operative procedures with any expectation of success. However, maintenance of blood supply, the avoidance of irritation during the process of healing, and complete release of tension of the flap and suture lines are necessary requirements for success with any flap, wherever cut or placed.

The genitals have an abundant blood supply. With ordinary care in the construction and placement of the flap, its life can be maintained even though flaps are cut in reverse to the channels of nourishment. Yet loss of tissue seems, according to the literature, to be a major consideration in replacing flap operations with other plans.

Fig. 9.—The picture of the condition on Nov. 2, 1938. A completely released organ, an intact urinary channel, and so far no complications. The flap has accommodated itself to a 14 F caliber channel. In this case the bladder cannot be entered by a flexible catheter, because a pouch was formed which lies ventrally and a little below the opening made by the umbrella flap. It is necessary that a rigid catheter, hugging the dorsal wall of the channel, be used. Note a balanitic hypospadias.

The avoidance of irritation in the process of healing is a general surgical principle in the care of any wound, but here it is most important because the whole effort of the operation is to construct an avenue of evacuation. Therefore it is necessary to divert the urinary stream by suprapubic, perineal, or catheter drainage.

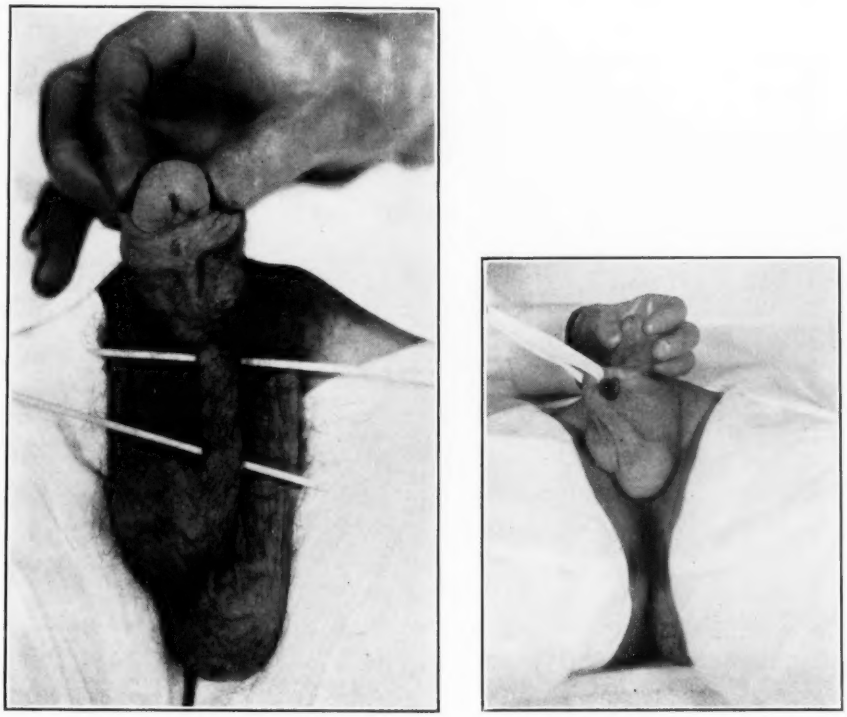
The complete release of tension on flap and suture lines is another general surgical principle widely discussed in wound repair. In this situation this requirement is elevated to the peak of consideration, because several factors must be overcome to meet this requirement.

The parts cannot be immobilized. The genitals are appendages to the trunk and cannot be firmly sealed by dressings and support as is possible in other body areas.

The operation is done on a movable base. This same situation occurs in the soft palate, on the lip, and in the tissues about the orbit and

possibly in some of the muscle tendon transplants. But here there is no possibility of anticipating the frequency or degree of motion, due to the erectility of the penis.

It is impossible to anticipate the degree of postoperative edema. The scrotal tissues are more prone to swell after surgical attack than any other tissues of the body, except possibly the orbital tissues. Every surgeon will recognize that this factor has a very important bearing on the question of stitch tension.



A. *B.*

Fig. 10.—The third case shows a midpenile hypospadias, a degree amenable to repair by the Duplay-Thiersch, Cecil, Browne combination. Had this been done in adolescent life, hair would have developed in the tube, because in this picture hair appears on the penile tissues up to the level of the opening. *A*, Note the profusion of hair on the flap and visualize the condition in any case wherein scrotal tissues are transplanted without preparation. *B*, The condition after six months of preparation. Compare this flap with the one shown in Fig. 6*A*. The flap in this case is too narrow and has given me some delay in release because of a coalescence of the puncture wounds at the base. The flap is viable and, although two fractional amputation incisions have been made, it has not caught up with its return blood supply. There is no reason to handicap oneself because of the wide spread of the tissues of the scrotum. The wider the plot, the longer the flap can be made at the first sitting.

There is limitation of the spread of the epithelial envelope of the penis. These unchangeable factors which make the avoidance of tension on suture lines so difficult doubtless explain the reason why the problem of repair of hypospadias apparentiy has been a great challenge over so many years.

My main difficulty in the repair of hypospadias has been in the limited spread of epithelial envelope of the penis in the operations, which

I personally have accepted as the proper method of approach. In these operations there is a dearth of tissue to meet the question of stitch tension. On the contrary, with the use of the scrotal flap, a limitless supply of tissue is afforded and the problem of stitch tension is likely to be met with greater assurance of success.

Let us consider the penile flap and scrotal flap operations separately.

PENILE FLAP OPERATIONS

The need in operation for the repair of hypospadias is the construction of a tube in the position of a normal urethra and of the same length and caliber. The operation that best meets this requirement is a tube flap, cut from the ventrum of the penis, sutured in reverse,

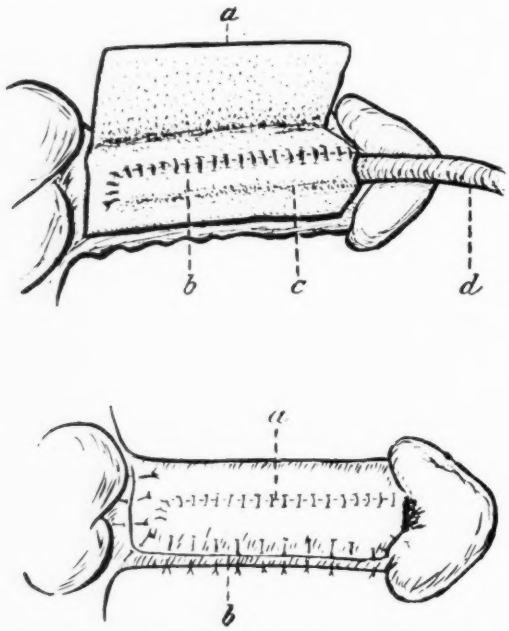


Fig. 11.—The Duplay-Thiersch-Cecil combination of flaps, the ideal operation for repair of penile hypospadias, to be done following the suggestion of Browne as shown in Fig. 12. (From Browne: An Operation for Hypospadias, Lancet 1: 141, 1936.)

and buried under the lateral sliding flaps, according to the now classical Duplay-Thiersch technique. If this operation were always successful, there would be no need for further consideration or discussion. Why has this method been put aside in favor of so many diverse methods? The reason is that not infrequently a fistula or fistulas or a complete breakdown of the suture lines have followed. The most frequent explanation of partial or complete failure is infection. Yet these tissues have a high degree of immunity and in more recent years there have been developed a number of very effective nonirritating antiseptics. Another explanation may be that unwittingly, in the suture of the tube flap, the needle has entered the channel, making an avenue for the

formation of a fistula. But is it not much more reasonable to attribute failure most often to tension on the suture lines, produced by erection, plus the tendency of these tissues to swell?

The difficulty I have had in the Duplay-Thiersch operation is in exactly plotting the tube flap. If the flap is made too generous, surely tension on the outside suture line follows. If it is made too narrow, it then becomes necessary to suture the thin margins of the flap in mere contact in an awkward position.

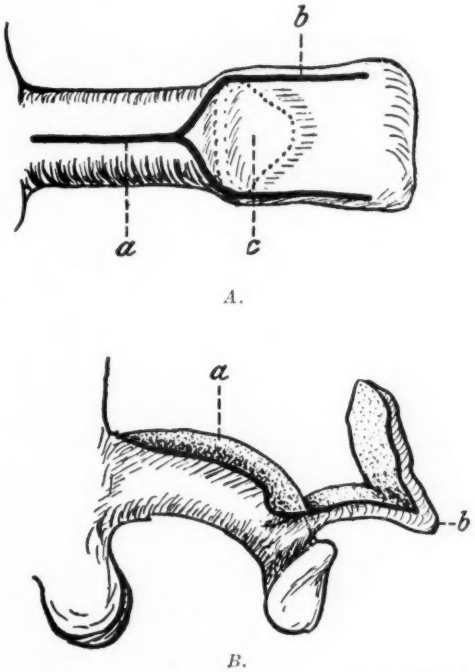


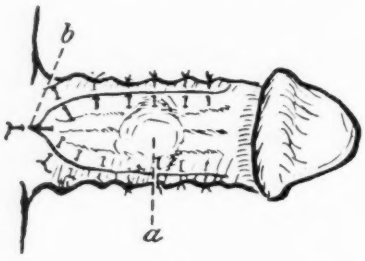
Fig. 12.—The plan of Browne to increase the spread of the epithelial envelope of the penis by receding a single-ended pedicle flap of the prepuce. Note that this flap is cut in reverse of the blood supply which comes from the base of the penis. *A*, The plot; *B*, the flap. (From Browne: An Operation for Hypospadias, Lancet **1**: 141, 1936.)

Cecil modified the Duplay-Thiersch technique by a suggestion, in the cutting of flaps, of staggering the suture lines of the inner tube flap and the lateral sliding flap, the flaps being cut short on one side and long on the other (Fig. 11). From the viewpoint of tissue transplant, this procedure is a conversion of the flaps of the Duplay-Thiersch operation into overfolding flaps. I believe that these flaps are built on the same surgical principles, are splendid contributions and now generally accepted by all surgeons. I personally accept the Cecil modification as a milestone in the solution of the problem of repair of hypospadias.

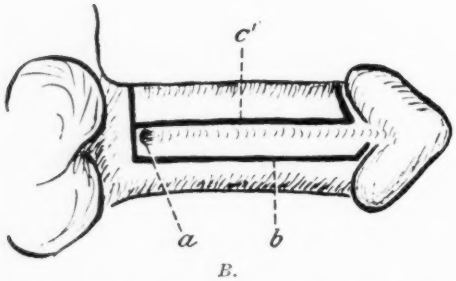
The technique of Duplay-Thiersch and the Cecil modification do not take cognizance of what I believe is the most important requirement of success, sufficient tissue to meet stitch tension. It is to be observed that the covering of the penis lies loose when this organ is relaxed

and snug when erect. Although the penile tissues are very elastic, the expectation that they will meet on erection two different suture lines, on two different flaps, assumes a grave risk of failure.

The limitation of tissue in the penile flap operations has been long recognized by Ombredonne in an operation which increases the spread of the tissue on the ventrum of the penis by bringing the prepuce forward in the form of a double-ended pedicle flap. The Ombredonne operation yields a high percentage of successful results and has been supported by many critical surgeons. The reason for this success is the greater amount of tissue made available to meet the several factors inimical to release of tension of the stitches.



A.



B.

Fig. 13.—*A*, The flap placed on the dorsum of the penis, the Browne modification. *B*, The staggered incisions on the ventrum of the penis, the Cecil modification. (From Browne: An Operation for Hypospadias, Lancet **1**: 141, 1936.)

Denis Browne now makes an original suggestion to overcome the handicap of insufficient tissue on the ventrum. The plan is original, not in the form of the flap but in its placement: This form of a single-ended pedicle flap is the oldest in surgery and is old in the repair of hypospadias, having been used as far back as the 1870's by Van Horn and in more recent years by Edmunds. It has been recommended in several modern contributions. But in all its previous usage, this flap was brought to the ventrum of the penis. Denis Browne places this flap on the dorsum. The situation can be described as a stem, around which is an epithelial envelope. The envelope is incised, the tissues mobilized to the ventrum, and then the defect is filled in by this flap cut from the prepuce (Fig. 12).

We have done this operation but once. Wallace Ritchie placed the flap with ease, with a perfect result, except for a spot of superficial necrosis along one of the suture lines, which healed readily. This flap is cut in reverse to the avenues of nourishment which come from the base of the penis. It requires care in preparation to meet the first requirement of flap transplants, maintenance of blood supply. By thus increasing the volume of the tissues of the envelope of the penis, a generous tube flap may be cut from the ventrum of the penis. Browne then suggests that this be sutured by the Connel intestinal stitch, which is, in effect, a continuous mattress, thus insuring a broad approximation of the denuded surfaces with a minimum danger of entering the channel by a stitch.

To illustrate this part of the paper, I have copied the illustrations of Denis Browne from his original article, because I believe that they illustrate by far the most reasonable combinations of flaps for the penile degree of hypospadias. Fundamentally, this is the Duplay-Thiersch operation, which purports to build a tube in the position of a normal urethra, plus the modification of Cecil, plus the modification of Browne.

With this combination of flaps as a basis of study, discussions can now be directed to the problems of technical details, sequence of, and age for operation.

SCROTAL FLAP OPERATIONS

The handicap of insufficient tissue to assure release of stitch tension has been recognized by the plan of Ombredonne, the plan of Browne, and also the plan of Gatewood who by his stepup operation takes cognizance of the growth and accommodation of the tissues, and also by a technique widely used in the transplant of tissues, whereby successive operations are employed instead of the attempt being made to complete the operation at one sitting.

A fourth plan to meet the handicap of insufficient tissue is the use of scrotal tissues. The scrotal tissues are redundant, far beyond the actual normal requirement of covering for the testes, and no penalty is paid for their use. They are plastic, pliable, vital, and can be handled to meet all the essential requirements of flap transplant with more assurance of success than any other tissue in the body.

This fact has been recognized in a number of operations which invade the scrotum. To discuss or compare them in this paper would be no more than a repetition of what has been so beautifully done in the literature and more recently in a masterly way by Godard. However, for the sake of my argument, I selected for comment the Bucknall operation, offered in 1907.

When the scrotal flap operations are lined up for comparison, it is interesting to note that no matter how the operative steps differ the operations all have one procedure in common: *The penis is buried in the scrotum.*

From the viewpoint of tissue transplants, this surgical step is now described as *carrying the deformity to the flap.*

I present a plan for the use of scrotal tissues based on the proposal to *prepare the flap and carry it to the deformity.*

The reason that the former plan has been always used in hypospadias is the fact that the penis when relaxed lies easily in contact with the scrotum and it is most reasonable to expect that this position would readily meet the question of stitch tension after the flaps are sutured.

In the Bucknall operation (Fig. 14) a flap is cut from the ventrum of the penis and sutured to a flap cut from the scrotum for the floor

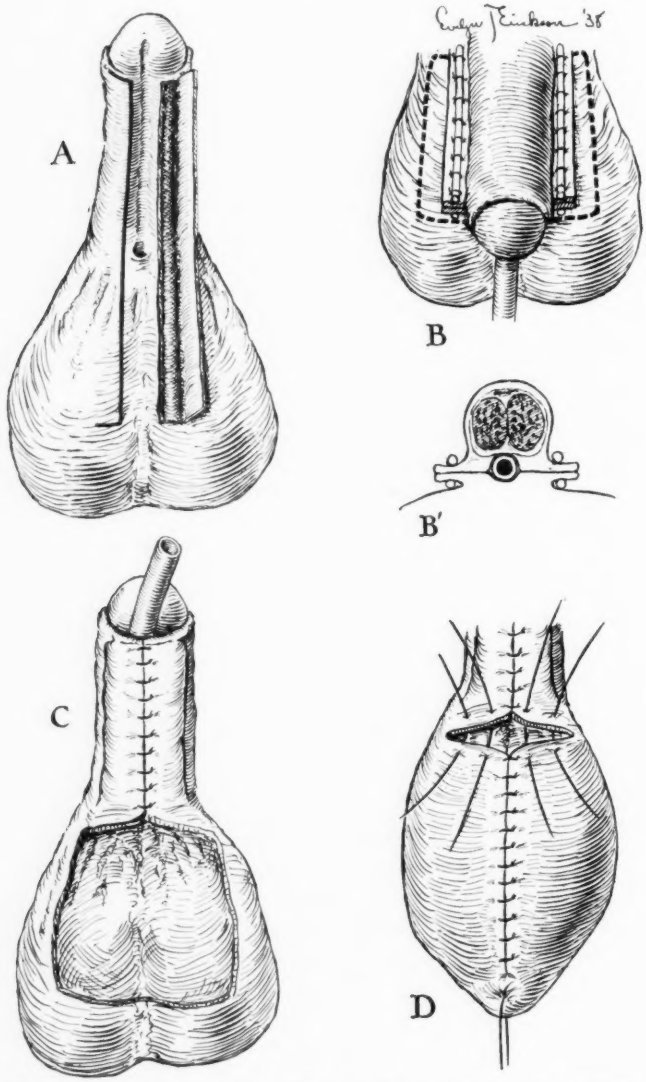


Fig. 14.—The Bucknall operation. It is here selected as an example of the use of scrotal tissue whereby the deformity is carried to the flap. It is to be compared with the plan for the use of scrotal tissue whereby the flap is prepared and carried to the deformity. (From Creevy: Operative Treatment of Hypospadias, SURGERY 3: 726, 1938.)

of the urethra. Thus two suture lines are buried and two exposed, four suture lines at one operation. Following a period of time to permit union, two more flaps, a right and left single-ended pedicle form, are cut wide of the suture lines to cover the released penis and the scrotal flap. Keen surgical judgment is required to determine the time of release. The main objection to this operation is that the second principle of flap transplant is emphasized and the two operations require a diversion of the urinary stream for an indeterminate period of hospitalization. Even with such care, cases are reported with illustrations showing points of failure. While some of the scrotal flaps recognize that the scrotal tissues are hair-bearing after puberty, many do not.

(At this point of the argument, to avoid repetition, will the reader please scan the legends of Figs. 1 through 10, not only to criticize the technical details and sequence of procedure there shown, but to compare the operation with the Bucknall, each an example of the use of scrotal tissues by two different methods of approach.)

When I saw hair growing in the channel following one of the steps in the repair of the case illustrated, I went over the literature again to find the status of the hair problem. It has been discussed from the beginning of the literature. I cannot find the exact reference, but the story is that some one suggested, when first the use of scrotal flaps was offered, that it must be expected that, should the case live past puberty, a beard would appear at the meatus. The objection is not so exaggerated because to emphasize this phase of the subject I will say that in the illustration the mat of hair seen in the channel had the appearance of young beard.

Bucknall discussed the hair problem. He suggested that the penile flap be made wide and the scrotal flap narrow, thus transplanting a minimum amount of scrotal tissue. He also wondered whether the follicles would develop in their new position. Of course, they will, because the main requirement for success in flap transplant is live tissue.

I think everyone interested in these problems should read the article by Churchman, because he brings together the many failures following the repair of this deformity and it is startling to find all the things that may happen. He vigorously supports the Bucknall technique as following a proper surgical principle, but it is interesting to note that no mention is made of the hair problem.

Articles in the recent literature overlook this phase of the subject.

Every year I see cases reported where a scrotal degree of hypospadias has been cured by the Ombredonne technique. I presume the reason for this lack of discussion of the hair problem is found in the present accepted ages for operation.

The general opinion is that the first operation for release of the penis from the contractural band may be done as a matter of convenience, depending upon the condition of the child. The requirement of this operation is that a complete release of encurvature is obtained.

The present idea is that hypospadias should be operated upon any time between the ages of 7 and 10 years, at least with a definite purpose to complete the case before puberty. At such an age there is no keen differentiation between the penile and scrotal tissues and, therefore, depending on the degree of the deformity, scrotal tissues may be included in the tube, whether penile or scrotal flaps are used.

In the reported case, I operated upon this patient at 14 years and 3 months of age, still appearing a boy. He returned to me at 15 years and 7 months of age, developed as a man. There is an occasional report of a case in which a hair ball has formed after puberty with deposit of lime salts, making what seems to me a serious complication at a time of life when this organ really begins to be of some use to the individual.

But these reports are few and far between, and from this experience I wonder if there are not a number of unreported cases of this complication. Is the formation of hair in the urethra of importance to the individual? Is it just as easy to take care of the hair formation after the operation? If so, what has been done? What is the relation of the hair question to the sex problem? These are questions I would like discussed, but they are, I believe, beyond the scope of my paper.

I would be less than frank were I to give the impression that the use of the scrotal tissue by this form flap and method of approach was developed through any initiative on my part. It was not, but it was used in my first case because of a peculiar set of circumstances.

CASE REPORT

A married man, 50 years of age, had lost, through some destructive inflammatory process some years previously, the whole urethra up to the penile-scrotal junction. The left cavernous body and one-half of the prepuce was the site of a hard, brawny scar. The hair question was not presented because the scrotal tissues were hairless, whether from the inflammatory process or long treatment, or both. I conceived the idea of building a tubed flap from both right and left scrotum and planned to swing them up on each side of the penis and sew them together. The left flap failed to meet the maintenance of blood supply in the flap transplant and an area of necrosis appeared in the midportion. During a period of uncertainty as to the next step, the right flap became flaccid, elastic, and evidently contained sufficient tissue to complete the repair. I brought it up first on the right side of the penis with catheter drainage of the bladder. In six months I completed by two series suture lines on the left side and in this instance diverted the urinary stream per perineal section. The result of these plans was most happy. The man assured me that I had constructed not only an intact urinary channel, but also a competent sexual organ.

This patient must be classed as a traumatic case, but the picture was identical with the penile-scrotal degree of hypospadias. The application of the procedure to the congenital deformity never crossed my mind, but when the problem arose, as shown in Fig. 4, I knew what to do.

The form of the flap is old in the literature but in recent years has been popularized by Gillies. It can be built to enormous lengths, providing the plot of it is properly made and the time element in its construction is correctly gauged. In this location, even in the most extreme case of perineal hypospadias, a flap can be made by encircling the scrotal tissue in different planes to fit the most extreme development of this organ. It is worth while to state that the flap remains rather rigid for an arbitrary period of three months, but gradually assumes the aspects of the normal scrotal tissues as shown in Fig. 7*B*.

I am uncertain as to the method for depilation. The objection to the fulguration needle is that it is difficult to control the area of destruction, with the possibility of coalescence and the formation of broad scar. This occurrence is of importance only at the base of the flap through which nourishment must come. The use of the x-ray as a depilatory agent is deprecated.

Possibly the treatment of depilation of the hair follicles can be better assured by excision. The hairs of the scrotum are discrete. When pulled by gentle traction, a white spot appears at the base. This can be anchored by a fine forceps and then clipped, which, of course, should be done in the long axis of the flap. Should a definite wound result, it may be stitched. Contiguous hairs can be taken care of at future sittings and thus overlapping wounds avoided. All of this may be done under local anesthesia without hospitalization.

It is to be noted (Fig. 9) that the end result in this case is a balanitic hypospadias. Is this sufficient? The literature so indicates and it was entirely satisfactory to my completed cases. However, should this result be unacceptable, the flap may be built long enough to extend beyond the glans.

I have wondered whether the flap so placed would withstand the traumatism of coitus. My first patient assured me that it would, but this is only one case. Possibly it would be better, in placing the flap, that a three instead of two series suture line be used. The fact is that with such a wealth of tissue available one may increase the spread of the covering of the penis to any degree selected.

In this case, because of my experience of a loss of a flap in my first case, I made a wide plot of the flap, thinking that at the second operation it could be cut to fit; but when the second line of sutures was made, the flap lay so evenly and easily without tension that it was used in toto. The result eighteen months later is that it has accommodated itself perfectly.

I am in no position vigorously to advocate the operation. I can say that there is no plan that I have used where I have been so free from concern during the process of healing. The patient voids, the parts are dried, protected by a diaper, the suture lines are exposed for care and observation, and the diversion of the urine required only for the last series suture lines. All of which is due to the fact that the quantity of tissue transplanted was sufficient to give not only a margin, but a wide margin, of safety, to meet the several factors inimical to stitch tension peculiar to this part of the body.

CONCLUSIONS

1. The crux of the problem of repair of hypospadias is to find sufficient tissue to guarantee release of tension on the suture lines.

2. A revision should be made of the questions of age for operation in order to meet the hair question.

3. The literature should be sifted of several procedures, which evidently were once suggested on the idea that something special should be attempted to repair the deformity.

4. The problem of repair of hypospadias should be approached from the viewpoint of tissue transplants, because the operative steps involved are familiar to every surgeon and have been discussed from the dawn of medical history.

5. The tissues used in the repair should be those of the genitals, because they are native to the parts and thus have the potentiality of future growth and accommodation in the new position.

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UNDESCENDED PERINEAL TESTIS

Report of a Case

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ECTOPIA testis is an unusual condition and ectopia testis perinei is therefore still more uncommon. "Potential" perineal testis, i.e., an undescended inguinal testicle associated with the anatomic features of perineal testis, is apparently rare. For that reason the following case is reported.

CASE REPORT

D. C. (University of Virginia Hospital No. 128889), a 13-year-old white boy, was admitted to the hospital on July 12, 1938, because of an undescended right testicle. An older brother had been operated upon for the same condition in 1932.

Examination revealed a well-developed boy with no testicle in the right scrotum. The penis was mature and the left testicle was somewhat larger than the average. The right half of the scrotum was small and its lateral attachment was closer to the midline than usual. Lateral to the right half of the scrotum and separated from it by a deep groove there was found an anomalous, labium-like fold of skin that extended from the region of the pubic spine to a point well down in the perineum (Fig. 1). Within the fold there was felt a linear structure thought to be the ductus deferens. The superficial inguinal ring on the right side was small and was identified with difficulty. In the position of the inguinal canal on the right there could be felt a rounded body that was approximately 1.5 cm. in diameter. There was an indirect inguinal hernia on the left side.

On July 13, 1938, a left inguinal herniorrhaphy was first done by the Ferguson method. The right inguinal canal was then opened and the processus vaginalis, a large sac communicating with the abdominal cavity, was immediately encountered. Projecting into the posterior wall of the sac was the right testicle, epididymis, and ductus deferens. The peritoneal sac extended downwards over the pubis into the labium-like fold in the perineum described above. Also extending into the fold was a loop of the ductus deferens and the gubernaculum testis. The latter was a well-developed structure whose proximal end was attached to the lower pole of the testis and whose distal end was firmly fixed to the bottom of the anomalous perineal fold.

The peritoneal sac was transfixed and ligated at a high level and amputated. The lower portion was then freed from the testicle and the structures of the cord and dissected out of the perineal fold. A finger was thrust into the right scrotum and a channel was easily burrowed out beneath the superficial fascia. There seemed to be a natural point of lessened resistance through which the finger passed. The gubernaculum was cut at its insertion in the perineal fold. The spermatic cord was of adequate length, and a typical Torek procedure was carried out without division of fascial bands or vessels.

PERINEAL TESTIS

There was found upon reviewing the literature some variation in the terminology used in referring to abnormalities of testicular de-

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scent. To avoid confusion, the terms maldescent or imperfect descent will be used to include all variations from the normal descent of the testis. The term ectopic testis will be used to mean a testicle that has descended into an abnormal position away from the course taken by the normal testis as it descends into the scrotum.

Although in rare instances perineal testis has been reported to occur as the result of trauma,[1] we are concerned in this discussion only with the anomaly of congenital perineal testis. The condition is not common. Altogether there have been recorded somewhat over 100 cases in the literature. Perineal testis was first described by John Hunter[11] in 1786, when he reported 2 cases. The first detailed account of the anomaly, however, was given in 1857 by Curling,[7] who collected 9 cases. In 1879 Annandale performed the first successful operative

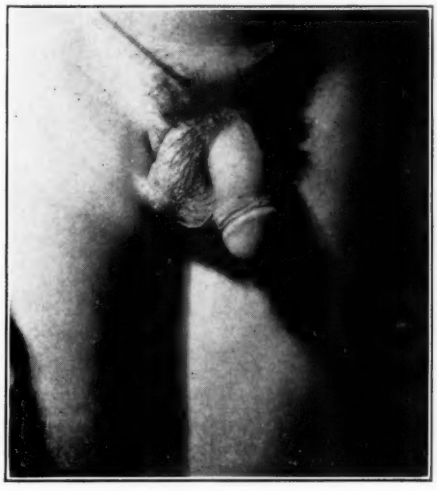

Fig. 1.—Case of undescended perineal testis. Photograph taken between the first and second stages of the Torek operation. Note just behind the right scrotum the labium-like fold of skin which contained the ectopic processus vaginalis and gubernaculum.

cure of a case of perineal testis.[5] By 1889 the reports of 30 cases had been collected by Monad and Terrillon.[16] In 1899 Weinberger[21] collected 74 cases, and in 1906 Klein[13] assembled 81.

In 1857 Godard[10] found 3 cases of perineal testis in a series of 53 cases of maldescended testis studied by him. In 1903 Eccles[9] found only 5 cases among 936 cases of imperfectly descended testis associated with hernia. W. B. Coley[5] says that from 1890 to 1907 at the Hospital for the Ruptured and Crippled there were 737 cases of maldescended testis associated with hernia and that in only 15 instances was a testis found in the perineum. All but 6 of the 737 cases came to operation. One hundred twenty-six of the cases were operated upon by Coley himself and 9 of these had a perineal testis. Burdick and B. L. Coley[3] report 537 operations for maldescended testis on 482 patients at the Hospital for the Ruptured and Crippled from 1891 to

1924. In this group there were 86 cases of ectopic testis, but not one of these was of the perineal type. Campbell[4] states that there was no case of perineal testis found among 36,000 urological admissions at Bellevue Hospital.

NORMAL DESCENT OF THE TESTIS

The details of the mechanism by which the testis normally becomes transferred from its position of origin on the dorsal abdominal wall to its final location in the scrotum are not entirely understood. During the fourth week of fetal life, the mesonephros begins to develop from the nephrogenic cord or unsegmented intermediate mesoderm in the dorsal portion of the embryo. Growth of the mesonephros causes it to bulge ventrally into the celomic cavity, where it is known as the urogenital fold. Cranialward the urogenital folds of the two sides are separate, but caudally they fuse together to form the genital cord. The celomic epithelium on the ventromedial surface of the urogenital fold becomes thickened and many layered, so that by the sixth week it is bulging independently into the coelom as a longitudinal fold, known as the genital fold, which is the primordium of the gonad. The genital fold increases in thickness and becomes constricted away from the mesonephros by deepening medial and lateral grooves. These grooves remain separated from each other by a thin attachment or mesentery which, in the case of the testis, is later to become the mesorchium. As development of the genital gland continues, the more cranial segments of the primordium degenerate so that it becomes shortened and more compact. The degenerating cephalic segments persist for a time as the diaphragmatic ligament which is attached to the cranial pole of the gonad.

Toward the end of the second month the component parts of the gubernaculum develop. The most caudal portion of the genital fold becomes fibromuscular and attaches the testis to the genital cord. It is known as the ligamentum testis. About the seventh week of fetal life there appears an inguinal fold that extends between the genital fold and a prominence on the anterior abdominal wall, called the inguinal crest. Within the inguinal fold there is developed a fibromuscular band called the chorda gubernaculi. The abdominal musculature becomes developed around the chorda to form the inguinal canal. At its distal end the chorda gubernaculi becomes connected to another band, the ligamentum scroti, which extends to the scrotal swelling and later to the bottom of the scrotal pouch. Now by condensation to form a fibrous cord within the genital cord, the proximal end of the chorda gubernaculi becomes united to the distal end of the ligamentum testis, thus effecting a continuous ligamentous structure, the gubernaculum, which extends all the way from the caudal pole of the testis to the scrotal swelling.

At the beginning of the third fetal month an evagination of the peritoneum takes place at the site of the future abdominal inguinal ring to form a saclike pouch, the processus vaginalis. This peritoneal process passes through the inguinal canal and over the pubis into the scrotum.

By the end of the third month the early degeneration of its cranial portion associated with a more rapid elongation of the trunk has produced a relative descent of the testis, so that it occupies a position at the boundary between the abdomen and the pelvis near the future abdominal inguinal ring. There is little further change in the position of the testis until the beginning of the seventh month, when the gubernaculum has ceased growing and has begun to shorten. Most anatomists believe that the further descent of the testis is brought about fundamentally by a traction force of the gubernaculum. The testis is pulled into the inguinal canal dorsal to the vaginal process of peritoneum. During this period the gubernaculum shortens by one-half. The gubernaculum continues its process of shortening and the testicle passes out through the superficial inguinal ring and finally reaches the scrotum, usually about the end of the ninth lunar month, or shortly before birth. In the newborn male the gubernaculum is only about a quarter of its original length. The proximal part of the processus vaginalis becomes obliterated just before or soon after birth, while the distal part persists as the tunica vaginalis testis.

While the degeneration of the cranial portion of the early gonad, the disproportionate growth of the trunk as compared to that of the genital gland, and the traction force of the gubernaculum are at present considered to be the main factors in the production of the descent of the testis, there are other forces which probably play a part to a greater or lesser degree. Intra-abdominal pressure, resulting from the growth and enlargement of the viscera of the abdominal cavity, probably helps to force the testicle into the inguinal canal in much the same manner that an inguinal hernia is formed. Active contraction of the abdominal musculature undoubtedly aids the testicle in its passage through the inguinal canal.

ETIOLOGY OF ECTOPIC TESTIS

In reports of cases of congenital ectopic testis there are numerous theories offered in explanation of the ectopia, many of which center on the gubernaculum.

A once popular gubernacular theory for the explanation of ectopic testis in general was advanced by Lockwood[14] in 1887. Lockwood thought of the gubernaculum as a structure with multitailed insertions. He considered one strand to go to the scrotum, one to the root of the penis, another to the perineum, another to the pubis in the region of the superficial inguinal ring, and finally one to the femoral

triangle. Lockwood, therefore, explained ectopic testis upon the basis of overdevelopment of a gubernacular strand attached in an ectopic region. This theory in recent times has been largely discarded.

Others, notably Sonneland,[18] think that ectopic testis is mainly contingent upon an insufficiency or rupture of the gubernaculum, which, in certain cases, may become reinserted in an abnormal location. Still others ascribe the cause of ectopia to a primarily abnormal insertion of the gubernaculum. Burdick and Coley[3] believe that certain types of ectopia are due to rupture of the gubernaculum and that other types, including perineal ectopia, are the result of an abnormal gubernacular insertion. Congenital insufficiency of the length of the spermatic cord is another explanation that has been offered.

Next in frequency are the obstructive theories of ectopic testis. These theories all assume some mechanical means by which the testicle is obstructed in its descent or pushed from its normal course. McGregor,[15] in a careful study of the fascial planes of the anterior abdominal wall and perineum, describes various congenital fascial pockets and fascial ridges that, when present and well developed, may interrupt or deflect the testicle in its descent. Other mechanical explanations for ectopia include such conditions as fat plugging the scrotum, hernia pushing the testis from its course, and adhesions developing between the testis and the neighboring tissues.

Uffreduzzi[20] considers heredity a very important element in the production of ectopic testis. The heredity theory was first advanced in 1857 by Godard,[10] who reported a case in which both a father and son had a perineal testicle. In twenty-four cases of ectopic testis examined by Uffreduzzi, other anomalies were found in each. Uffreduzzi claims that degenerative signs can be found in the great majority of the cases of ectopic testis.

COMMENT

The chief significance of the case here reported lies in the fact that both the gubernaculum testis and the processus vaginalis extended into a fascial pocket in the perineum. If the gubernaculum is the principal force that governs the descent of the testicle following its emergence from the inguinal canal—and this is the prevailing opinion—then it seems reasonably safe to say that if the patient's testicle had descended it would have descended into the perineum. Hence, it should be considered a "potential" or an "undescended" perineal testicle.

Coley[6] refers to this type of case when he states that in some cases of inguinal retention of the testis the processus vaginalis may extend beyond the inguinal canal to the scrotum but occasionally may miss the scrotum and pass into the perineum.

A perineal fold or pouch is not, apparently, an uncommon finding in cases of perineal testis. According to Sonneland[18] "a fascial pocket

or sac is a striking part of the picture'' in both perineal and penile positions of the testis. Describing his case of perineal testis, Robertson[17] speaks of a pocket in the perineum in which the testicle lay, being attached there by a gubernaculum 3 mm. in diameter. Pulling on this fibrous band, he says, would cause a dimpling of the skin of the perineum. Eccles[8] also speaks of an adventitious sac, in cases of perineal testis associated with hernia, that lies lateral to and is separated by a sulcus from an imperfectly developed scrotum.

The present case presents an interesting element of heredity in that the patient's brother had an operation for undescended testis at this hospital in 1932. In the brother's case the left testicle was found in the inguinal canal and the processus vaginalis had not descended beyond the testicle.

It is believed that this rare case offers some evidence bearing on the unsettled problems of abnormal descent of the testis. Sonneland[18] says: ''Congenital perineal testis is often accompanied by some abnormality of the corresponding vaginal process of peritoneum. No instance is known in which the vaginal process found its way into the scrotum while the testis went into the perineum. Since under normal conditions the vaginal process precedes the testis into the scrotum, it probably precedes the testis into the aberrant position in these abnormal cases.'' Sonneland's prediction is fulfilled in this case. It is evident, therefore, that there must be some common factor that is responsible for the combined aberration of both the processus vaginalis and the testicle in cases of perineal ectopia; otherwise both of these structures would not always be ectopic at the same time. Inasmuch as the processus vaginalis precedes the testicle in its descent, it might at first appear that the primary aberration is one of the processus vaginalis. However, when we consider that the gubernaculum is developed somewhat ahead of the descent of the processus and that the former does not descend but is developed in situ, so to speak, from three sources, it is difficult to see how the processus vaginalis could influence the position of the gubernaculum and, hence, the final position of the testicle.

The cause of the descent of the processus vaginalis and its direction normally into the scrotum is obscure. The peritoneal process probably originates as a result of increasing intra-abdominal pressure and is directed into the inguinal canal by the abdominal inguinal ring. It may be assisted through the canal by the contraction of the surrounding musculature, but after its emergence from the superficial inguinal ring there is no ligamentous structure present to pull or guide the processus into the scrotum. How it gets there is purely a matter of speculation. It may be that there is a plane of reduced tissue density that extends to the scrotum; or the gubernaculum, which lies posterior to the processus, may act as a growth-stimulating focus along which

the cells of the peritoneal pouch grow toward the scrotal sac. The gubernaculum cannot directly pull the processus vaginalis into the scrotum, because at this stage the two structures are not adherent. Furthermore, the gubernaculum does not begin to shorten until after the processus has reached the scrotum. Should the ligamentum scroti portion of the gubernaculum be developed in an abnormal position, lateral to the scrotum with its distal attachment in the perineum—possibly in the bottom of a supernumerary scrotal pouch—it is conceivable that the gubernaculum might mechanically block the passage of the processus vaginalis into the scrotum and deflect it also into the perineum. Such a mechanism could readily explain the combined aberration of both gubernaculum and processus vaginalis that occurred in the case here reported and may possibly be the etiologic basis of perineal testis. The anatomic characters of the present case, i.e., the fully developed perineal processus vaginalis and gubernaculum, together with the inguinal position of the testicle, offer evidence that a common cause has deflected the entire direction of development. Whether or not the primary element behind this deflection lies with an abnormal gubernacular origin must remain a matter of speculation.

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SUBCUTANEOUS DORSAL DIGITAL BURSITIS

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SUBCUTANEOUS adventitial bursae are known to occur about the bony prominences of most of the major joints of the body. Those of most frequent recognition when traumatized or diseased are the prepatellar and olecranon bursae. The dorsal digital bursae of the fingers are subject to disease with almost as great a frequency and may be the cause of continuing disability or prolonged medical treatment if they are unrecognized as the primary source of continued pathologic change in the finger.

Spalteholz[1] pictures and describes these small bursae lying between the skin and the extensor tendons on the dorsum of the finger joints as follows:

1. "Dorsal Digital Subcutaneous Bursae. Immediately beneath the skin, on the dorsal surface of the digital articulations, usually small and are fairly constant over the first interphalangeal joints, occasionally occur over the distal interphalangeal joints."

2. "Dorsal metacarpophalangeal bursae occasionally occur on the dorsal surface of the metacarpophalangeal articulations, and then usually only on the 5th finger."

3. "Inter-metacarpophalangeal bursae, of frequent occurrence, from one to three in number. Small, and lying between two neighboring fingers, at the metacarpal heads dorsal to the transverse capitular ligaments, between the second and 5th fingers."

A particularly good illustration in Toldt[2] shows these bursae clearly.

In 1933 a minor finger wound on the dorsum of the index interphalangeal joint under treatment in the office healed, broke down and drained, healed, then drained repeatedly. A foreign body, bone, or joint infection could not be made out. A glance at the illustration of Spalteholz brought out forcibly certain points in the anatomy, forgotten since student days. When the roof of the previously unrecognized bursa was removed, allowing complete drainage, prompt and lasting healing occurred. This first case was under treatment for fifty days. Since then, twenty-six further cases of digital bursitis have been recognized in office patients.

The usual history is that, following a cut, abrasion, or scratch on the dorsum of one of the finger joints, the small wound heals. A latent period of from three to twenty days is followed by increasing soreness of the joint, swelling, redness, and tenderness. The wound may reopen and drain pus or the inflamed bursa may rupture and

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drain beside the healed scar. With rupture, relief of symptoms lasts only a few days. Healing, reappearance of inflammatory signs occur, and the cycle is repeated. With added new trauma, local cellulitis, lymphangitis, and adenitis may appear. The bursitis is recognized by swelling of the dorsal surface of the joint, tenderness only on the dorsum, not on the lateral or palmar surfaces, and pain on flexion which limits the motion of the finger. If one unroofs the bursa with a sharp scalpel, leaving only the floor and shallow walls, prompt healing takes place (Fig. 1). In cases recognized early or in more acute infections developing before the wound lips are healed firmly, spreading the original wound wide apart and keeping it open allows healing by secondary intention in a short time.

Fig. 1.—Acute dorsal digital subcutaneous bursitis over distal joint of fifth finger. The bursa was unroofed with a sharp scalpel five days before photograph. The exposed bursa appears as the central depressed area, its walls no longer sharp and steep and with healing under way.

The series of bursae here reported measures 3 by 3 mm. in average diameters. After unroofing them, they are easily recognized as a punched out crater, 3 or 4 mm. deep. The lining membrane is glistening gray without visible slough or fibrin. The wound secretion is viscid, clear, or purulent. These bursae were found most frequently in young adults (average age, 30.1 years) and more often in men (24 cases) than in women (3 cases). While butchers, fishermen, and warehousemen form the majority of these patients, housewives or stenographers may develop digital bursitis.

The metacarpophalangeal bursae are the most frequent sites of bursitis of the fingers. Thirteen metacarpophalangeal bursitis cases were observed, 3 of which had simultaneous infection of the adjoining

intermetacarpal phalangeal bursae. Intermetacarpal phalangeal bursitis is recognized by pain on lateral compression of the metacarpal heads and the depth of the drainage, signs of true purulent arthritis being absent. Proximal interphalangeal or midjoint bursitis occurred in 9 instances, and the bursae over the distal phalangeal joint were inflamed in 5 cases. It is evident that the metacarpophalangeal joints suffer trauma more frequently. The individual fingers show the following frequency of bursitis; mid- or third fingers, 12 cases; index finger, 9 cases; fifth finger, 3 cases; ring finger, 2 cases; thumb, 1 case. Spreading cellulitis was a complication in 5 cases.

Acute traumatic hemorrhagic bursitis was observed in 2 patients following blunt nonpenetrating trauma. Splinting the affected finger for a week proved effective, nondisabling treatment. One instance of chronic metacarpophalangeal bursitis was observed on the midfinger knuckle of a file clerk who pushed back heavy files with the dorsum of the affected hand.

In 21 patients the bursitis was recognized early in the course of treatment. These patients averaged 13.4 days of treatment and 2.1 days of disability. Two patients with acute cellulitis are included in this first group and were disabled 17 and 23 days respectively. Six patients received treatment from other physicians before coming to the office or were seen in consultation. An average of 74 days had elapsed before the bursitis was recognized. This second group averaged 44.1 days away from work. The average time necessary for permanent healing after unroofing the bursae was 22 days. The average total time of these patients under a physician's care was 92 days. Such cases are probably the source of medical and folk lore, concerning poor healing over the knuckles, "The wound keeps breaking open." Keep it open and it heals more quickly.

From 1933 to 1938, 22 cases of acute subdeltoid bursitis (peritendinitis calcificans), 27 instances of traumatic prepatellar bursitis, and 20 cases of acute olecranon bursitis were seen in office patients. The relative frequency of digital bursitis, and the patent's economic loss, the prolonged treatment of insignificant wounds, when such bursae are unrecognized, call for more vigilance in the treatment of minor finger injuries.

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TRANSCOLONIC REMOVAL OF POLYPS

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THE present study is concerned with those cases of polyposis of the colon in which the polyps were removed surgically through a small incision in the colon. Only those cases were selected in which the polyps were limited to a small segment of the colon and in which they were situated too high in the bowel to be removed through a proctoscope or sigmoidoscope. The clinical aspects, laboratory data, surgical treatment, pathologic observations, and results of treatment as judged by follow-up studies five or more years after operation are considered.

CLINICAL ASPECTS

Of the 62 patients studied, 31 were males and 31, females. The average age of the patients was 50.7 years. The oldest patient was 71 years of age and the youngest, 10 years of age.

The outstanding presenting symptom in these cases was bleeding from the rectum; 57 of the 62 patients complained of this symptom. The bleeding was usually intermittent and at times amounted to as much as 1 or 2 ounces of bright or dark blood. The patients often stated that bleeding occurred after a bowel movement. The average duration of the bleeding was thirty-three months. In one case bleeding from the rectum had occurred for ten years.

Symptoms other than bleeding from the rectum were not characteristic of these cases. Eleven patients complained of diarrhea. Constipation occurred in 12 cases. Four patients complained of alternating diarrhea and constipation. Twenty-eight patients suffered abdominal distress which varied widely in situation, character, and severity. In 2 cases bouts of severe abdominal cramps were associated with intussusception. One patient who at operation was found to have a polyp of an intussuscepting type did not experience abdominal distress.

LABORATORY DATA

Despite the long duration of the bleeding from the rectum, the average concentration of hemoglobin in these cases was 14 gm. per 100 c.c. of blood. The value for hemoglobin in the case in which the most severe degree of anemia was found was 8 gm. per 100 c.c. of blood.

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A proctoscopic examination was made in 58 cases. In 36 of these cases the polyps which were removed surgically were seen on proctoscopic examination. In each case the proctologist stated that the polyp could not be removed safely through the proctoscope. In 21 cases the proctologist fulgurated polyps in the rectosigmoid or rectum.

A satisfactory roentgenoscopic examination was conducted in 56 cases. The polyps were discovered in 50 cases. The 6 patients among whom the roentgenologist failed to obtain evidence of polyps were examined before a satisfactory roentgenographic technique for demonstrating polyps of the colon was developed.

SURGICAL TREATMENT

Three days prior to operation the patient was hospitalized. Preoperative preparation consisted in the administration of a nonresidue, high carbohydrate diet, colonic irrigations twice daily, sodium phosphate orally and vaccine intraperitoneally. At operation the diseased portion of the colon, as indicated by the roentgenoscopic and proctoscopic examinations, was exposed and the polyp was located by palpation. In cases in which the polyp was very small, or in cases in which the patient was obese and epiploic tags felt like polyps, the lesion was located by transillumination of the colon with the Cameron light. When the polyp was identified definitely, the diseased portion of the colon was isolated between the rubber-covered blades of a curved clamp. This portion of the colon was packed off carefully from the remainder of the peritoneal cavity. Usually the colon was incised in a longitudinal band for a distance of about 2 cm. The polyp was delivered from the colon. The stalk of the polyp was crushed by a forceps, the base was ligated, and the polyp was excised by using either a knife or cautery. The wound in the colon was closed with two rows of chromic catgut sutures. Epiploic tags, when available, were tied over the line of suture. In an occasional case in which the possibility of soiling the peritoneal cavity existed, a soft rubber tube was employed as a drain. Postoperatively fluids were administered parenterally until the patient passed gas by rectum, at which time the patient was permitted to drink fluids. A rectal tube was inserted frequently to facilitate the passage of gas. The patient was gradually permitted to eat a nonresidue diet which was cautiously replaced by a normal diet.

PATHOLOGY

A single polyp was removed in 57 cases. Two polyps were removed in each of 4 cases and in 1 case four polyps were removed. The polyps were situated in the sigmoid in 42 cases, in the descending colon in 15 cases, in the transverse colon in 3 cases, and in the cecum in 3 cases.

One patient had polyps in the transverse colon as well as in the sigmoid. In 39 cases the polyps were malignant. Thirty-one of the malignant polyps were graded according to the method of Broders. Twenty-seven were Grade 1 and 4 were Grade 2 (on a basis of 1 to 4).

RESULTS OF TREATMENT

There were 2 postoperative deaths or a surgical mortality rate of 3.2 per cent. The cause of death in one case was peritonitis and in the other case pulmonary embolus and bronchopneumonia.

The postoperative course of 19 patients was noted for periods varying between five and eighteen years. The living patients numbered 14, of whom 5 had malignant lesions and 9 had benign lesions. Among 11 of the living patients a more detailed follow-up study was possible. Eight of these patients were well, had not required further operation, and had not noted bleeding from the rectum since operation. In 3 cases further bleeding from the rectum had occurred. One of these patients was re-examined at the clinic. Roentgenoscopic and proctoscopic examinations failed to reveal the source of the bleeding. This patient was advised to return frequently for examination. Another patient who had had further bleeding from the rectum had a peptic ulcer after a posterior gastroenterostomy had been performed and the possibility of the presence of a bleeding jejunal ulcer was not excluded.

Five patients were dead. Only 1 of the patients who were not living had originally a benign lesion. One patient whose original polyp was malignant and was situated in the sigmoid returned to the clinic three years after his operation and on surgical exploration he was found to have carcinoma of the rectosigmoid with extensive metastasis to the liver. This patient died a few months later. Another patient whose lesion was malignant returned to the clinic one year after her operation and when the surgeon opened her abdomen he found a carcinoma at the junction of the cecum and ascending colon. He stated that undoubtedly the lesion had developed from a polyp. Metastasis had occurred to the liver. The original polyps were removed from the transverse colon and sigmoid. Either the polyp in the ascending colon was not present or was not discovered at the time of the first operation. The patient died three years after her primary operation.

One patient whose polyp was malignant died of myocarditis. Another patient from whom a malignant polyp had been removed was reported dead of an unknown cause.

The only patient not living whose lesion was benign died after surgical exploration of the abdomen in a distant city. A tumor of unknown origin was found in the liver.

COMMENT

The cases presented are a highly selected group. Most of the solitary polyps of the colon are low enough in the bowel to be removed through the proctoscope. During the past decade the proctoscopist has increased his field of operation by employing careful technique and the cases in which polyps of the colon must be removed abdominally have been proportionately fewer than in previous decades.

The patients studied were usually of middle age; representation of the sexes was about equal. Bleeding from the rectum was the only characteristic symptom presented by these patients. The long duration of the bleeding was striking; one patient had been bleeding from the rectum for ten years. It is equally striking that despite the long duration of their bleeding these patients had only slightly reduced values for hemoglobin. Cessation of bleeding from the rectum offered an index of cure after operation; in 8 of 11 cases further bleeding from the rectum did not occur after the polyps had been removed.

Roentgenoscopic and proctoscopic examinations were two of the most important means of diagnosing polyps of the colon. As Buie has stated, during the past ten years the accuracy of both of these examinations has increased greatly. The proctologist's advantage lay in the fact that he was able to view the polyps directly. Since, in the cases studied, polyps were situated too high in the colon to be removed safely through the proctoscope, it is remarkable that the proctologist was able to see 62 per cent of the polyps which were removed.

The roentgenoscopic examination was a particularly valuable diagnostic measure in the present series of cases because the polyps were situated near or beyond the reach of the proctoscope. The 6 patients, among whom the roentgenologist did not discover the polyps, were all examined prior to the use of the air insufflation technique of Weber. Buie cited a case to illustrate the accuracy of the roentgenoscopic examination. A patient whose proctoscopic examination was negative was found by the roentgenologist to have several small polyps in the colon. On re-examination the proctologist found and fulgurated the polyps. The roentgenologist then was not able to find more polyps in the colon.

The roentgenoscopic and proctoscopic examinations were the most important means of determining whether the disease had been eradicated. That such a decision may be very difficult to make was shown by the case in which rectal bleeding continued after the operation and neither the roentgenologist nor the proctologist was able to demonstrate disease in the intestinal tract.

The fact that in 39 of the 62 cases malignant polyps were removed indicates the danger involved in keeping under observation a patient who has polyps of the colon. Although the malignant lesion was of a

low grade, that it was dangerous was shown by the fact that two patients returned to the clinic and were found to have a malignant lesion and metastasis.

Compared with other surgical procedures, transcolonic removal of polyps is a relatively rare operation for malignancy of the colon. A sufficient number of cases have not been studied to determine whether or not this procedure is sufficiently extensive for use in dealing with malignant polyps of the colon. It was encouraging that 14 of the 19 patients whose postoperative course was followed for five years or more were living. Nine of the 14 patients, however, had benign polyps removed. In only 2 of the 5 cases which terminated fatally could one be sure that the cause of death was malignancy of the colon. In the case of the patient who returned to the clinic three years after his operation and was found to have extensive malignant lesions at the site of the original polyp, a more extensive surgical procedure might have yielded a better result.

CONCLUSIONS

A very small number of solitary polyps of the lower portion of the colon cannot be removed by the proctoscopist and must be removed by a transcolonic procedure. The risk of the operation, which in the present series of 62 cases was 3.2 per cent, is justified because in 39 of the 62 cases the polyps were malignant. Of the 19 patients whose course was followed for five to eighteen years after operation, 14 were living. Of the 5 not living, it is certain that 2 died of malignant lesions of the colon.

Bleeding from the rectum, which was the most characteristic symptom presented by the patient, is also valuable as an indication of cure after operation. Roentgenoscopic and proctoscopic examinations, the most valuable aids in the diagnosis of polyps of the colon, are necessary to determine whether or not the disease has been eradicated.

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A NEW CLAMP FOR THE DEVINE COLOSTOMY

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UNDOUBTEDLY, one of the most important advances that has been made in surgery of the large bowel is the preliminary performance of a new type of colostomy devised and advocated recently by Sir Hugh Devine.[1] Indeed so significant is this procedure that it may even be considered revolutionary. Its obvious rationale and perfectly logical conception are immediately apparent. Based on the principle that a completely isolated segment of bowel which is thus deprived of its function loses most of its bacterial content in a relatively short period of time, the colostomy is performed with the purpose of preparing an involved segment of the colon for its subsequent safe extirpation. The procedure, by "defunctioning the distal colon," actually converts a highly dangerous operative area teeming with pathogenic microorganisms into a relatively sterile and safe one. We are so impressed with its surgical rationale and fundamental advantages that it has become a routine preliminary procedure on the Tulane Surgical Service in these cases.

A more detailed account of the principles, technique, and indication of the procedure will not be discussed here, as this is presented in another publication in which is also given our experience in twenty-six cases[2] and a modification of the operation.

In brief the colostomy is performed by making a long spur (8 to 12 cm.) in a loop of the colon proximal to the involved area, dividing the bowel, and inserting the two ends in separate openings in the abdominal wall. Thus the proximal and distal colostomy openings are completely separated or "disconnected" by approximately 4 cm. of skin surface. The fecal stream is completely diverted and spillage into the distal segment is absolutely obviated. Moreover, by frequent lavage of this isolated segment through the distal colostomy opening, the "debacterilization" process can be materially enhanced. Thus the resection can be performed in a practically sterile field.

After the surgical procedure in the involved segment has been completed and healing has occurred, continuity of the bowel is re-established. Devine accomplishes this by a specially devised enterotome with elbowlike curves between the crushing blades and the handle. The blades are separately introduced in the respective colostomy openings, connected by a pivot screw, and, as the handles are gradually drawn together by a wing nut, the crushing blades approximate each

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other. In a few days the blades have cut through the long spur between the proximal and distal limbs of the bowel and continuity is re-established.

This type of clamp has been used in our cases and has been found to have certain disadvantages. It is somewhat bulky and inconvenient. Because of its weight and the long handles which project above the skin surface, it tends to fall on its side unless supported by a specially constructed bandage. For the same reason it causes discomfort and pain, probably because of traction on the mesentery.

Ideally the clamp should be so constructed as to possess lightness in weight, simplicity in design, facility in application, and convenience in operation. Moreover, it should create a graduated, evenly distributed pressure along its crushing surfaces on the spur. With this objective in mind, we have devised a clamp which is believed to fulfill these desiderata.

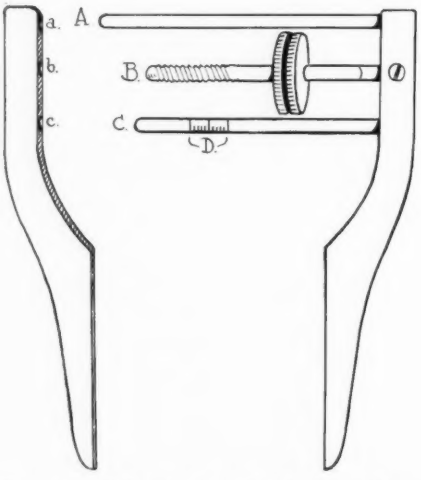

Fig. 1.—Drawing of the two parts of clamp disassembled.

The clamp is constructed of duraluminum and consists of two somewhat S-shaped arms (Fig. 1). The upper parts of these arms serve as handles for the clamp and the crushing surface which is longitudinally grooved is on the lower part. Two right angled crossbars (Fig. 1 *A* and *C*) are rigidly attached to the handle of one arm of the clamp. Corresponding openings (Fig. 1 *a* and *c*) are present in the other arm of the clamp through which these crossbars slide. Between these two crossbars is a screw provided with a wheel (Fig. 1 *B*) and also firmly attached to the arm of the clamp but so arranged that it turns freely. A corresponding screw opening is present in the other arm of the clamp (Fig. 1 *b*).

The technique of application is extremely simple. The two blades of the clamp are greased and separately introduced into the respective colostomy openings. It will be observed from the illustration that the upper crossbar, the lower crossbar, and the screw bar are of respec-

tively shorter lengths. This facilitates assembly. The upper crossbar is fitted first into its corresponding opening in the other arm of the clamp which it then slides along until the lower crossbar meets its corresponding opening. This arm of the clamp then slides along both crossbars of the other arm of the clamp until the middle screw bar meets its corresponding opening and by turning the wheel the two arms of the clamp are drawn toward each other. Also as the screw wheel is turned, the crushing blades approximate each other until they gradually cut through the spur (Fig. 2). The lower crossbar is so calibrated that it is possible to determine at a glance the exact distance between the two crushing blades beneath the skin surface (Figs. 1, *D*, and 2).

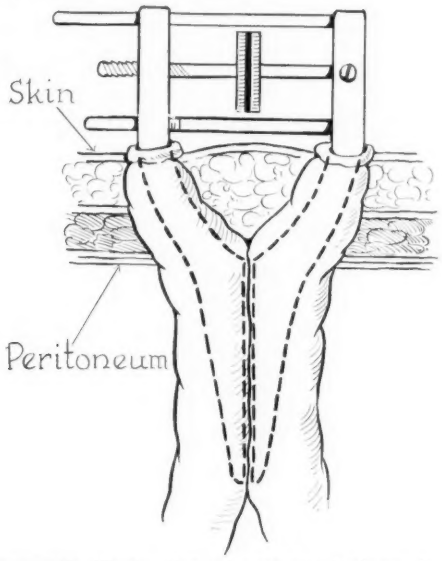

Fig. 2.—Diagrammatic illustration of assembled clamp in the colostomy.

The instrument is constructed of duraluminum to give lightness in weight. As the clamp consists of only two working parts, assembly and disassembly are considerably facilitated. The two crossbars assure stability, smoothness of operation, and an evenly distributed crushing surface. The crushing force is gradually and easily applied by the wheel screw. It can also be readily determined and constantly controlled by the calibrations on the lower crossbar. Because of its lightness in weight and the fact that the handles project only about 3 or 4 cm. above the skin surface, it is extremely convenient, simply requiring an ordinary dressing. Thus it causes no inconveniences or discomfort and the patient can be walking about while it is applied.

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Editorial

What Causes Hunner Ulcer?

OF ALL the urologic conditions of unknown cause, such as urogenital cancer, prostatic hyperplasia, polycystic kidneys, urinary lithiasis, and so on, the etiology of Hunner ulcer (submucous fibrosis) is the most evasive and annoying. Cancer of the urogenital tract is but part of a big general problem; ignorance of its cause is fully shared and the solution universally sought. The cause of benign enlargements of various portions of the prostate gland in more than one-third of all men over 50 years of age may be definitely related to certain hormonal changes occurring only in this proportion of our male population, or may be analogous to uterine fibroids with a similar basis of origin, but neither cause is particularly mystifying. Polycystic kidneys arise obviously from some developmental mishap, and the mental exercise of choosing, among the many theories offered in explanation, that one most applicable somehow appeases the urge for knowing the truth. Even more intelligible are the metabolic theories of stone formation and, while all the facts of causation and all the steps of formation of both polycystic disease and urinary lithiasis await discovery, enough is known to blunt one's curiosity. About other conditions of unknown cause, even those for which one guess is as good as another, someone generally comes close enough to satisfy ordinary inquisitiveness. The pathogenesis of Hunner ulcer, however, continues to be a mystery.

Too frequently Hunner ulcer is overlooked by the cystoscopist, the condition being completely unrecognized. Just as often, because of some supposed resemblance, he mistakes its identity and confuses it with some other condition. The diagnosis is purely clinical and can easily be missed or muddled. Only when uncomplicated are the symptoms pathognomonic and as a rule the visual picture is typical only before treatment. Then, frequency with pain, day and night, a clear urine, nothing more, point to a Hunner ulcer and, on looking in, a narrow line of inflammation is found on the dome of the bladder or at the side above one or the other ureteral orifice or, when extensive, arching clear across from above one orifice to above the other; a line of inflammation characterized by a seemingly intact mucosa which bleeds on the least vesical distention. The urine may show a few red blood cells but usually, before cystoscopy, is clear both macroscopically and microscopically, and no growth is found on culture. Even cultures of tissue from the inflamed area (taken by cystoscopic rongeur and at operation) generally show neither aerobes nor anaerobes. That

the lesion is caused by some organism yet to be discovered seems improbable. Pathologists variously describe submucous inflammation, an infiltration with small round cells with certain changes in the connective tissue. These descriptions are not always identifying and diagnostic, yet they differ from those of other types of cystitis and vesical ulcer.

Here then is an entity with unique symptoms and a definite cystoscopic picture. Therapeutic experience only has been added to our clinical knowledge of the condition since Hunner clearly described it. We know that recurrence usually follows surgical resection, that overdistention is beneficial, and that, when combined with fulguration persistently and regularly, it is curative. We do not know what causes the lesion nor why it responds favorably to this form of treatment. Its origin, production, and development remain as obscure and baffling as when first called elusive by Hunner, although he meant only cystoscopic elusiveness.

Why does Hunner ulcer affect mostly women and why mostly those between the ages of 30 and 40 years? Why are all males and the younger and older females relatively unsusceptible? Why is it so deep rooted and enduring, so chronic and malignant in nature? Why is it so characteristically linear and always so uniformly located? The trigone and vesical neck are never involved. Why not? Is anatomic structure at the bottom of the distribution of lesions and, if so, in what relation? Is it some perivesical factor, such as peritoneal reflection, or some intramural agency related to the line of wear of movable on fixed parts with filling and emptying? (We have often noted that the early lesions of submucous fibrosis seem to follow the line of flexure on itself which the bladder wall makes when it empties and fills.) If position signifies anything, is this anatomic basis of etiology then peritoneal, muscular, arterial, venous, lymphatic, or nervous? Is the lesion infectious, trophic, allergic or what? What possibly can cause it? Nobody knows.

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

RECENT ADVANCES IN THE MORE COMMON PROBLEMS OF MINOR ANORECTAL SURGERY

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THE treatment of anorectal disease too long remained in the hands of the charlatan, who glibly promised cures without surgery and dispensed "painless pile dissolvents." To be sure, for many years there have been occasional physicians and surgeons who have labored earnestly in this field, but it is only in the past few decades that any considerable number have concentrated upon the advancement of the understanding of its peculiar problems. Today this advance has attained considerable momentum, and anorectal surgery has achieved a conspicuous place as a specialization in surgery. The modern proctologist is fundamentally a surgeon well trained in the general principles of surgical diagnosis and technique. The vast majority of pathologic conditions occurring in this region must be approached and treated by the special application of general surgical principles.

No advance in such a special field of surgery is possible without increased understanding of the pathologic changes which can and do take place in the region involved. Great progress has been made possible in this respect by increasing general recognition of the importance of complete and careful examination of every patient who presents himself to the physician with any anorectal complaint. It is unfortunately still true that from 26 to 50 per cent of patients with carcinoma of the rectum, within easy reach of the examining finger, still receive treatment for some other supposed rectal condition for weeks and months before the correct diagnosis is made. But the importance of proper examination is now so stressed in medical education that such a record in the future will be inexcusable.

There has been in recent years a tremendous advance in the diminution of suffering which all too often accompanied any anorectal operation. Many new anesthetic agents have been developed to minimize the patient's postoperative discomfort. But of equal importance has been the intelligent application of certain principles in the technique of the operation itself: avoidance of undue trauma, avoidance of cutaneous sutures, and adequate surgical drainage of all wounds well

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out beyond the anus. Today anorectal operations can be approached both by the patient and by the surgeon with no greater apprehension of suffering than that which may accompany any other surgical procedure.

At the present time it would appear that the application of ambulant treatment to anorectal disorders is overstressed. To be sure, great advances have been made in the office management of minor surgical procedures, chiefly by the utilization of newer methods of prolonged anesthesia. This is in many instances justifiable and praiseworthy. But the result is all too often inadequate surgery with resultant delay in proper alleviation of the condition.

The medical literature of today contains annually hundreds of articles on diseases of the anorectal region. To undertake a review of the advances in all phases of the subject is beyond the scope of this paper. Attention has therefore been directed to a discussion of certain common disorders. Conflicting opinions naturally exist in a field which is served by the general surgeon and by the proctologist who may not have had the benefit of general surgical training. Personal opinion undoubtedly influences such a review, but this is unavoidable.

ANESTHESIA

Complete muscular relaxation is essential for efficient anorectal surgery. Relaxation of the sphincter and levator ani muscles allows the anal canal to open up and evert, resulting in proper exposure with an absolute minimum of pulling and retraction of tissues—a cardinal principle in this field of surgery. The anal sphincters are the last muscles of the body to relax under any form of inhalation anesthesia and will adequately relax only under deep ether saturation. In addition to the primary hazards of such an anesthetic there are further disadvantages. The frequent resulting nausea and vomiting produce violent distortion of the anal canal which may tear out sutures, displace dressings, and induce hemorrhage. Ether anesthesia also tends to be followed by spasmodic contractions of the anal sphincters[7] with an increase of postoperative discomfort. There is also variable delay in the early resumption of ingestion of fluids and solid foods. Happily, the use of general inhalation anesthesia for anorectal operations has been largely discarded. Nitrous oxide or cyclopropane anesthesia is adequate and desirable for incision and drainage of perianal or perirectal abscesses where no attempt is to be made to investigate or to operate within the anal canal. It may be used also for the occasional highly neurotic individual for analgesia, utilizing some other type of anesthetic for complete muscular relaxation.

The use of *intravenous anesthetics* has achieved a certain popularity, principally for procedures that can be completed within five or ten minutes. Evipal, introduced in 1932,[186] and pentothal sodium, intro-

duced in 1935,[112] are the agents of choice. Their action is characterized by ease of administration, rapidity of induction of deep anesthesia, complete muscular relaxation, short duration of anesthesia, prompt recovery, adequate margin of safety, and rare after-effects.[47, 81, 82, 111, 112] Smoother action, less noticeable fall in blood pressure, quicker recovery, less subsequent amnesia are claimed for pentothal sodium by Haskell,[81] who has found anesthesia satisfactory in 85 per cent of 175 anorectal operations requiring less than ten minutes of anesthesia. Attention has been called[47, 82] to the variable period of amnesia following anesthesia lasting thirty minutes to twelve hours. This must be seriously considered in the application of this method of anesthesia to office procedures. Technique of administration is adequately detailed by Lundy,[111] Bacon,[7] and Haskell.[81] Contraindications for its use are hepatic disease, low blood pressure, any condition which produces gross interference with gaseous interchange in the lungs, idiosyncrasy to barbiturates, and extreme youth or old age.[7, 47, 82] Haskell and Cheleden[82] have collected 32 fatalities from the literature in several hundred thousand administrations.

The usefulness of this form of anesthesia would appear to be definitely limited to procedures requiring but a few minutes and necessitating good sphincter relaxation, such as the control of hemorrhage, incision of abscess, or removal of fecal impaction.

Regional Anesthesia.—Anesthesia with complete muscular relaxation is best achieved by interruption of the sensory and motor pathways at one point or another along the course of the nerves from the spinal cord to the terminal nerve fibrils.

Local infiltration, "free injection and saturation of tissues immediately adjacent to and beneath the field of operation or just proximal to the sensory nerve endings,"[7] is undesirable[28] except for the excision of a thrombosed external hemorrhoid, the removal of a papilla, or the trimming of cutaneous tags. There are two major objections: resultant deformity of the tissues and the danger of subsequent infection from contiguous inflammation.

Conduction anesthesia, or blocking of the conducting pathways proximal to the terminal nerve distribution, is a popular method of anesthesia. Although this is frequently referred to as local anesthesia, it differs from local infiltration in that the anesthetic solution is injected deep to the sphincter muscles without deformity of the more superficial tissues and with a minimal danger of subsequent infection. It is applicable in most minor anorectal operations with the notable exception of fistulectomy. Here the hidden possibility of extension of infection into the region of infiltration is a definite hazard and contraindication to its use. The technique of conduction anesthesia is described in detail by Bacon.[7] Introduction of 15 to 20 c.c. of anesthetic solution is made posteriorly through a point 1½ inches back of the

anus in the midline in three planes, one backward in the midline deep and paralleling the anal canal for a distance of 1½ inches, and one on either side deeply forward and outward 2½ to 3 inches. Similarly, through a point 1 inch anterior to the anus in the midline, the needle is passed upward 1½ inches parallel to the anal canal in the midline and then on either side deeply backward and outward 2½ inches, injecting a total of 15 to 20 c.c. of the anesthetic solution. A gloved finger is kept in the rectum during all of this procedure to be certain that the needle at no time pierces the anal skin or the mucous membrane above. The posterior injection serves to block the posterior branches of the inferior hemorrhoidal nerves, the anococcygeal nerves, and the perineal branch of the fourth sacral nerves. The anterior injection serves to block the posterior scrotal nerve, the lateral terminal branches of the perineal nerve, and the anterior branches of the inferior hemorrhoidal nerves. Complete relaxation and anesthesia ensue.

Caudal anesthesia has not been widely used in anorectal surgery because of its variable effectiveness, with failure of satisfactory anesthesia and relaxation in from 4 to 25 per cent of cases reported.[13, 14, 78, 146, 160] The theoretical advantages are those of a nicely controlled level of anesthesia, which is extradural, with none of the undesirable reactions occasionally associated with spinal or intradural anesthesia. Renewed interest recently has been shown in this method by Reuther,[147] who had no failures using 2 per cent metycaine, although he had had 55 per cent of failures using freshly prepared alkaline procaine.

Sacral block anesthesia, by combined caudal and transsacral block, has been proved to be a safe and effective method in the hands of those experienced in its use. This method has long been advocated by Buie,[30] who after 15,000 cases considers it the ideal anesthesia. Failure of its universal acceptance as the ideal anesthetic is essentially due to the fact that the method is time consuming, requires patience and the skill born of experience on the part of the anesthetist, and is more trying to the patient than the single introduction of a lumbar puncture needle. The method is used today by relatively few surgeons, although its adequacy and safety have for many years been demonstrated in the department of proctology at the Mayo Clinic. Block anesthesia of the sacral nerves as described by Buie[28] consists in the injection of 25 c.c. of 1 per cent procaine with 1 per cent of a 1:2,600 solution of epinephrine into the caudal canal, followed by injection of 15, 10, and 3 c.c. of the same solution respectively into the second, third, and fourth sacral foramina on either side. Anesthesia is to be expected in ten or fifteen minutes, will be intense in twenty to thirty minutes, and will last one to two hours.

Spinal anesthesia is considered by many to be the anesthetic of choice, producing prompt anesthesia and extreme relaxation with minimal discomfort to the patient in the course of induction. Improved

technique and the refinement of drugs employed has resulted in greater safety than in general anesthesia.[22, 53, 59] The alarming fall in blood pressure so frequently encountered in the use of high spinal anesthesia does not occur if it is restricted to the lower lumbar and sacral levels.[4] Diffusion of the anesthetic agent to high levels is dependent principally on the effect of gravity. Control of the level of anesthesia can therefore be satisfactorily accomplished by proper adjustment in the position of the patient, the head and shoulders being kept below the level of the hips when an anesthetic agent lighter than spinal fluid is employed or slightly above this level when the anesthetic agent is heavier than spinal fluid. An increased frequency of urinary retention is said to follow the use of spinal anesthesia.[7] This complication, however, is more closely correlated to the intensity of postoperative pain and the degree of sphincter spasm than to the type of anesthesia employed. The one real and annoying complication is "postspinal" or "meningeal" headache, which has been variously estimated[49, 99, 136, 178] as occurring in from 4 to 32 per cent of cases. Such headaches are not noted in patients who remain in bed for a week or more. But the average patient, following a minor anorectal operation, is allowed out of bed on the second, third, or fourth day and may promptly develop headache after he has been up. It is controllable by prompt return to the horizontal position and usually does not recur after two or three days. The occasional persistence of this symptom for one or two weeks can be the source of considerable annoyance to the patient and embarrassment to the surgeon. Although disputable, such headaches would appear to be associated with the leakage of cerebrospinal fluid through the needle puncture in the dura. It is notable that the patient who tosses himself about in bed and insists upon raising his head and shoulders and even sitting up in bed is much more apt to develop headache than the patient who lies quietly flat in bed for twenty-four to thirty-six hours. Some time ago I became discouraged with the use of spinal anesthesia because of the frequency of postspinal headache, although I considered this type of anesthesia as otherwise ideal. However, since the employment of a 23 gauge lumbar puncture needle and insistence upon strict horizontal position of the patient for thirty-six hours following operation, this annoying complication has practically vanished. Relief of postspinal headache is said to be achieved by pilocarpin sweats,[54] pituitrin or caffeine citrate,[7] intravenous caffeine sodium benzoate,[122] or 0.5 per cent solution of nitroglycerin in alcohol.[16] Nausea and vomiting have been noted in 5 per cent[59] to 15 per cent[98] of cases having spinal anesthesia. This is more prone to occur in nervous and apprehensive individuals and occurs practically always in women. The administration of morphine preoperatively appears often to be a factor in this condition and other medication is recommended, preferably one of the barbiturates.

Lumbar epidural or *peridural anesthesia* has received no particular attention as yet for anorectal surgery but in the future may prove to be highly satisfactory. Although the idea of epidural anesthesia was conceived in 1885 by Corning[41] and used in 1920 by Pagés,[142] a satisfactory method of localizing the peridural space was not discovered until 1928[85] and its usefulness not recognized until 1932, when Gutiérrez[72] enthusiastically reported its use in over 3,000 patients. Further favorable reports have been made by Dogliotti,[45] Harger,[77] and Odom.[141] This type of anesthetic is comparable to caudal and transsacral block anesthesia accomplished by a single needle puncture in the lumbar region, but without the possible complications of intradural spinal anesthesia. The hazard of this method lies in inadvertent puncture of the dura and possible injection of 20 or more c.c. of novocaine solution into the spinal canal.

Anesthetic Agents.—A tremendous enthusiasm is evinced in the literature of the past several years for the use of anesthetic agents which have the property of prolonged action for the reduction of postoperative discomfort. Before discussing the numerous agents that have been used for this purpose, it is worth pausing to consider critically the utilization of such measures. Great strides have been made in the reduction of postoperative pain by improvements in preoperative and postoperative care, the now almost universal elimination of the large rectal retention tube or "whistle tube," and above all by appropriate surgical technique performed under adequate anesthesia and relaxation. In not a few hands these measures alone have been entirely satisfactory in effecting a uniformly comfortable postoperative course. Too free use of anesthetizing agents of prolonged action must not be stressed to the exclusion of superior surgical technique. That this occurs is obvious in scores of published reports in which the importance of the particular anesthetic agent used overshadows the importance of the surgical procedure employed. Most of the solutions advocated are definitely tissue irritants; many of them have produced, at least occasionally, actual necrosis. Such measures violate one of the cardinal principles of anorectal surgery; namely, the avoidance of all undue trauma, physical, thermal, and chemical.[125] The danger of complicating infection as the result of the injection of a slowly absorbed solution contiguous to wounds which are often primarily and always secondarily infected must always be considered. There is, on the other hand, no excuse for universal condemnation of measures which may be of real assistance in the reduction of postoperative sphincter spasm and discomfort. Provided the hazards and pitfalls of such measures are always kept in mind, there is unquestionably a use for such agents. Great progress has already been made and further progress is to be expected in this field.

The most commonly used anesthetic agent for all types of regional anesthesia is novocaine or its prototype, procaine. For local infiltration and conduction anesthesia a 0.5 to 1 per cent solution is employed. For caudal and transsacral block a 1 to 4 per cent solution is used. For spinal anesthesia 25 to 60 mg. dissolved in 1 to 3 c.c. of fluid is customary. No anesthetic agent has yet been developed that is more effective, prompter in action, or less toxic. The rapidity of cessation of anesthesia is as great as its induction, regardless of the method of administration employed. This often is followed immediately by a period of hypersensitivity and spasmodic contractions of the sphincter ani muscles. Patients in whom the subsequent postoperative course is entirely comfortable may suffer considerable discomfort during a variable period of time shortly after their return from the operating room. It has been thought that the utilization of an anesthetic agent, the effects of which wore off more gradually over a longer period of time, might eliminate this disadvantage.

Quinine and urea hydrochloride was popularized for its prolonged anesthetic action early in the twentieth century. It was abandoned by the general surgeon because of the resulting induration, edema, and sometimes sloughing, but it is still used by some in anorectal surgery.[87, 188] Originally used in a 5 per cent solution,[67, 69, 93, 192] it subsequently was advocated in a 0.25 to 1 per cent strength.[157, 158] Saphir, after extolling its use as the "ideal local anesthetic"[157] for hemorrhoidectomy in over 2,000 cases, abandoned it[159] because of the immediate production of fibrin, delay in healing, and sloughing of tissues and returned to 0.5 per cent novocaine with 2½ minims of adrenalin to the ounce. In any effective strength it tends to cause considerable pain on injection, analgesia is slow to appear (5 to 20 minutes), and it is definitely irritating, producing variable degrees of tissue reaction with not infrequent necrosis and slough.[104, 159] The anesthetic action is said to last from two to ten days.[157, 188]

Quinine hydrochloride and urethane has been found by Kilbourne[104] to be a less painful and less irritating solution than quinine and urea hydrochloride.

Eucupin, a quinoline derivative, in 1:1,000 dilution in combination with 0.5 per cent procaine, was advocated by de Takats[44] in 1926 for its prolonged action and gradual cessation of analgesia. Manheim and Marks[115] in 1938 reported reliable anesthetic action for minor anorectal surgical procedures with eucupin dihydrochloride 0.1 per cent combined with procaine hydrochloride 1 per cent in saline solution, producing postoperative analgesia of three to seven days. Eucupin, 0.75 per cent in distilled water, has been found by Kilbourne[104] to relieve postoperative pain by topical application in wet dressings applied directly to anorectal wounds immediately following operation.

Nupercaine hydrochlorid, or percaine, a quinoline derivative, is considered by some[107, 183] to be more toxic than cocaine and ten times more potent. Bacon,[7] after using it in 91 cases for conduction anesthesia, reports results superior to novocaine: injection painless, prompt anesthesia lasting 6 to 6½ hours, no local irritation, and no untoward effects; 1:1,500 dilution is recommended. Keyes and McLellan[103] report analgesia lasting 6 to 8 hours. Jones[100] recommends percaine 1:1,500 for spinal analgesia.

Diothane, a derivative of phenyl urethane, was suggested by Rider[151] for prolonged anesthesia in 1930. This authority concludes[150] that when used subcutaneously it approximates procaine in toxicity, is relatively stable to heat and can be sterilized by boiling, has considerable bacteriostatic action, but must be prepared and kept in nonalkaline containers (pyrex glass) to avoid precipitation of free base. After careful study in 100 cases, Rosser[154] is of the opinion that 0.5 of 1 per cent diothane in an isotonic solution produces relatively little irritation and gives anesthesia lasting from several hours to several days. Because of the immediate discomfort upon injection, he suggests combination with procaine for infiltration anesthesia for such minor procedures as the excision of a thrombosed external hemorrhoid. He has found it to be very efficacious in the reduction of postoperative sphincter spasm and discomfort by conduction block immediately following hemorrhoidectomy under spinal anesthesia. It should never be used as a spinal anesthetic agent because of an apparent chemical incompatibility with spinal fluid. Smith reports[169] that a 1 or 0.5 of 1 per cent solution diffuses slowly in the tissues and must be distributed carefully to prevent pooling and possible slough. Anesthesia is slow (10 to 15 minutes). He finds definite decrease in postoperative discomfort by conduction block with 5 to 10 c.c. of 0.5 of 1 per cent solution immediately following operation under some other form of primary anesthesia. Bacon,[7] using 5 c.c. of 0.5 of 1 per cent solution of diothane for posterior conduction block immediately following operation under spinal anesthesia, reports an average duration of anesthesia of 90 hours in 118 patients. These patients had varying degrees of discomfort but no severe pain. Other favorable reports on the use of diothane have been made by Terrell,[180] Simmons,[164] and Hertzler.[88]

Tutocaine hydrochloride is considered to be twice as potent as procaine hydrochloride and one-half as toxic. Kilbourne[104] recommends its use as a 2.5 per cent solution for prolonged anesthesia.

Anesthetic Agents in Oil.—Numerous anesthetic agents are now in use in combination with sweet almond oil for prolonged anesthetic effects. This action depends primarily on the slow rate of absorption of the injected material. In contrast to the water-soluble anesthetic agents of prolonged action with analgesia lasting from several hours to several days, the effect of these preparations in oil is supposed to

last from several days to several weeks. Originally devised for use in treatment of fissure-in-ano and pruritus ani, their use has been extended by some to the control of postoperative pain. These solutions produce variable irritative reactions and must be used with caution. Injection of the solutions at body temperature is recommended to minimize reaction. Some[34, 70, 125, 130, 145] consider that there is no indication for their use in anorectal surgery.

Benacol, introduced in 1927 by Yeomans and co-workers,[194] consists of para-amino-benzoyl (ethacaine), 5 per cent; phenmethylol, 5 per cent; rectified sweet almond oil, 90 per cent. The use of this agent in the control of postoperative pain is emphasized by Hayes.[84] Infiltration is attended by burning pain which may last for several hours.

Gabriel's solution or A. B. A., introduced as an inexpensive substitute for benacol in 1929,[57] consists of anesthesin, 3 per cent; benzyl alcohol, 5 per cent; ether, 10 per cent in sterilized almond oil. Simmons[164] considers this the best of the oil-soluble anesthetics for the control of pain following hemorrhoidectomy. The use of this solution is not without discomfort to the patient.

Morgan's solution, introduced in 1935,[131] consists of procaine base, 1.5 per cent; butyl-para-aminobenzoate, 6 per cent; benzyl alcohol, 5 per cent; in sterile almond oil. Morgan claims painless induction if injected slowly, no severe afterpain, certain and prolonged anesthesia for 7 to 28 days, used in amounts up to 20 to 30 c.c. Reuther[148] has used this solution for anesthesia in minor anal operations, 10 c.c. producing no reaction, with skin anesthesia in five minutes. He found the minimal duration of anesthesia to be 5 days, the maximal duration 23 days, and the average duration 12 days.

Anucaine, introduced by Gorsch[66] in 1934, consists of benzocaine, 5 per cent; phenmethylol, 5 per cent; butyl aminobenzoate, 1 per cent; novocaine base, 0.125 per cent; in sweet almond oil. In a later article[63] this authority does not recommend the solution as an operative anesthetic but suggests its use to alleviate postoperative pain. In 3,000 injections, using 5 c.c. in each posterior quadrant and 2 c.c. in each anterior quadrant, he has experienced no slough and no general reactions of any severity, but in several cases there has been transitory induration in the perianal tissues or an urticarial rash in patients presumably sensitive to almond oil. He frequently injects the solution one to two days before operation.[62] Green[70] has made a carefully controlled study of the efficacy of anucaine by estimating the amount of immediate postoperative medication necessary, the frequency of postoperative catheterization, and the spontaneity of the first bowel movement after operation. He concludes that pain is definitely minimized by providing prolonged relaxation of the sphincter muscles. In regard to frequency of complications by its use he reports reactions due to drug sensitivity (prolonged sensory anesthesia, constitutional and local re-

action, itching and local urticaria) in 3.04 per cent, and reaction due to technique of injection (superficial necrosis and deep abscess) in 1.17 per cent of cases. In the majority of cases he has used 5 to 10 c.c. of the solution for supplementary anesthesia, but in some it has been employed for primary anesthesia.

Neothesol, composed of procaine base, 1 per cent; methyl methylene para-amino-phenylformate, 2 per cent; hydroxbenzocarbinol, 5 per cent; in refined French almond oil, is considered superior by Burt and Rennie.[32] He considers 15 to 20 c.c. adequate for most operations and 25 c.c. sufficient for hemorrhoidectomy, subcutaneous circumanal injection giving complete anesthesia of the skin. In a series of 194 cases, 6 per cent developed necrosis of tissue; 2 had generalized reactions.

Nupercaine base, 1.5 per cent; benzyl alcohol, 10 per cent; phenol, 1 per cent; in sterile sweet almond oil is recommended by Steinberg.[174]

HEMORRHOIDS

It is the deliberate intention of the author to omit from this review all discussion of etiology, predisposing factors, incidence, descriptive anatomy, classification, symptomatology, and diagnosis of hemorrhoids. These subjects are thoroughly discussed in standard textbooks on the subject. The introductory paragraphs of the chapter on hemorrhoids in Buie's *Practical Proctology* are recommended for reading to anyone interested in the subject of treatment of hemorrhoids.

There has been increasing recognition of the fact that true appreciation of the presence and size of hemorrhoids cannot be gained by mere external inspection, nor is digital examination of particular value because of the softness and easy compressibility of internal hemorrhoids. Internal inspection is of prime importance, preferably with an anoscope which has an oblique internal orifice. It has also been increasingly recognized that patients do not actually suffer from hemorrhoids but from the complications of hemorrhoids, and proper treatment must depend upon what those complications are. In the determination of treatment it is important to recognize internal and external hemorrhoids as such. Those situated above the mucocutaneous or pectinate line and covered with mucous membrane are always internal hemorrhoids and those situated below this line and covered by skin are always external hemorrhoids regardless of their relative position inside or outside of the anal canal at the time of examination. All too often the doctor as well as the patient attempts by main force to "reduce" an acutely thrombosed external hemorrhoid, inflicting more harm than good upon the unhappy patient. In the attempt to correlate the symptoms and the physical findings for the determination of proper treatment, it is important to remember that the mucocutaneous line is also the landmark for the upward extent of sensory

innervation. When the patient's chief complaint is pain, the cause of this pain must be at or distal to this line and regardless of how big or how vascular or how eroded are his internal hemorrhoids it must be realized that this pain is not going to be relieved by treatment directed solely to the internal hemorrhoids. It is also important to remember that, regardless of how large the hemorrhoids may be, the patient's symptoms may not be from the hemorrhoids at all but may be due to a great variety of other causes, including cancer. Careful and complete digital, anoscopic, and proctoscopic examination should always be performed and a satisfactory cause for all symptoms found, to the exclusion of other possible causes, before any treatment at all is instituted for the hemorrhoids.

In the consideration of the numerous recommended methods of treatment for hemorrhoids, it must be emphasized again that the only satisfactory application of any of these methods will depend upon the surgeon's complete understanding of the true pathologic condition present and the correct application of the proper method of treatment to the particular condition existing.

Thrombosed Hemorrhoids.—Thrombosis of external hemorrhoids was long considered to be the result of a mechanical rupture of an anal varicosity with formation of an extraluminar hematoma. Such diffusion of blood in the perianal tissues does occur, but is rare in comparison with the frequency of multisacculated intraluminar thromboses which may result from a tiny tear in the intima of a thin-walled vein but which are probably frequently secondary to infection in the region of the adjacent crypts. Conservative treatment of the small thrombosed external hemorrhoid by rest, hot Sitz baths, and stool lubrication will be followed by gradual resolution and fibrosis in the course of from a few days to a week or more. Decision as to surgical intervention rests upon the judgment of the surgeon and it may well be employed if it is felt that pain and incapacity can be definitely reduced by such a procedure. Incision and expression of the clot are to be discouraged as this rarely shortens the natural course of the disease. Block excision of the multisacculated clots should be done through an elliptical incision in the overlying skin so devised that the edges of the incision will fall accurately together at the end of the operation. If properly accomplished, with no sutures or ties employed, relief is practically complete in less than twenty-four hours.

The advisability of immediate operation in cases of diffuse thrombosis of external and internal hemorrhoids with associated edema, infection, and necrosis is considered questionable. Cartwright[35] discusses the subject thoroughly and analyzes 116 returns on a questionnaire sent to members of the American Proctologic Society. Immediate operation was favored by 76 and 38 considered it inadvisable. Those who practice delay do so because of fear of embolism, liver abscess,

or stenosis as the result of inability to judge the amount of tissue necessary to remove. Cartwright concludes that the danger of embolism and liver abscess is greater without than with operation which is performed under block anesthesia without clamps and without anal divulsion. Lockhart-Mummery[109] favors immediate operation and states that he has never seen any ill effects from such procedures.

Hemorrhoidectomy.—Careful perusal of the literature of the past few years does not lead to the impression that the operation of hemorrhoidectomy has yet escaped from its unenviable reputation for postoperative suffering. The various "types" of operation in vogue a hundred years ago all still have their proponents. The exaltation of "ambulant methods" is frequently justified by reference to the prolonged suffering incurred by operation. And yet in actual fact perhaps the greatest advance in "minor" anorectal surgery has been in the operative treatment of hemorrhoids. This has not occurred as the result of a new "type" of operation but by awakening recognition of the fact that hemorrhoidectomy is fundamentally a plastic operation performed upon tissues of peculiar anatomic characteristics in a region incapable of complete physiologic rest and inevitably subject to infection. This principle has been eloquently expressed by N. D. Smith in an article entitled "Hemorrhoidectomy: A Plastic Operation."[171] By the application of this principle Newman[140] finds that only 2 per cent of patients following hemorrhoidectomy require a narcotic and then morphine sulfate gr. $\frac{1}{6}$ once only. Merar,[125] in 1939, lays down certain rules for hemorrhoidectomy: (1) Avoidance of every form of injury, mechanical, thermal, chemical, (2) application of that expert and particular care to which this area, as one of the most sensitive regions in the human body, is entitled; (3) utilization of a tried and tested routine preoperative and postoperative regime; (4) selection of an open, nonburning, nontraumatizing method of operation. Buie in his book *Practical Proctology*[28] says: "We dispense with formulas and urge that no particular type of operation be applied to all cases . . . rarely is it possible to do any series of operation in the same identical manner. We must adapt ourselves to conditions . . ."

In plastic surgery great emphasis is placed on preoperative preparation and particularly on postoperative care of wounds. This is of vital importance in hemorrhoidectomy and upon this depends in considerable part the postoperative comfort of the patient. Merar[125] emphasizes the importance of preoperative preparation to offset postoperative gas pains and to relieve the patient of any desire to defecate for at least three postoperative days. He recommends 6 ounces of citrate of magnesia twenty-four hours prior to operation, hospitalization the night before operation, with a plain water enema that night followed by a retention enema of 4 ounces of olive oil, to be expelled in the morning and followed by another plain water enema. For a

restful night to reduce preanesthetic fear and psychic trauma, he recommends 3 to 6 gr. of sodium amytal, giving another 3 gr. on awakening and dilaudid $\frac{1}{20}$ gr. one hour before operation. In regard to postoperative care Smith[171] stresses the importance of gentle cleanliness, the frequent use of nonirritating antiseptics, and the generous application of heat. Frequent hot Sitz baths should be started not later than the third day for cleansing, relaxation of the sphincters, and their soothing and healing effects upon the wounds. There are two noteworthy omissions in present-day postoperative routine: The large retention rectal tube routinely employed a few years ago, and the practice of "tying up the bowels" for several days to a week or more after operation. The "whistle tube" was a constant source of irritation to the wounds and elicited a reflex spasmodic contraction of the sphincters which was harmful to healing and most uncomfortable to the patient. Gentle dilatation of the anal canal is recognized as desirable a few days after operation to prevent adherence of raw surfaces within the canal and to keep the wounds open for proper distal drainage. Dilatation by the insertion of a finger is painful, largely because of the resistance of the sphincter muscles. Such resistance, however, is minimized by the natural coordination of these muscles in the normal physiologic act of defecation. Therefore the early passage of a soft but formed stool will accomplish the desired effect with the minimum of discomfort. To this end it is recommended that the patient be placed upon a regular diet immediately after operation and be given some agar-agar or tragacanth preparation to assure a formed but soft stool. Spontaneous defecation will usually occur on the third or possibly fourth postoperative day and is far more desirable than the use of enemas or more drastic catharsis. Patients may be dismissed from the hospital on the day following the first spontaneous bowel movement. Examination should be done at least twice weekly to be sure that proper healing occurs. On about the eighth day it is desirable gently to introduce the finger into the canal to be certain that spontaneous dilation by bowel movements has been adequate.

The importance of complete muscular relaxation for correct plastic surgery cannot be overstressed. This has already been discussed under anesthesia. The prone ventral position with the hips slightly elevated is recommended[28] to facilitate easy exposure and to provide an unhampered position for the surgeon. The objective of operation is thorough removal of the groups of varicose veins underlying the mucous membrane of the distal portion of the rectum and the skin of the anal canal with satisfactory functional result and a minimal deformity.[171] By careful technique and proper regard for sound principles in the care of infected wounds, all of the hemorrhoids can be removed without producing undesirable distortion or disturbing the function of the parts. Following a properly performed hemorrhoidec-

tomy, hemorrhoids will not recur and it is only when all hemorrhoids are not removed that they do recur.[28] It is important to recognize the fact that sterilization of the operative field is impossible. Therefore all unnecessary tissue damage must be avoided, all tissue which may slough must be removed, and adequate surgical drainage must be provided for.[171] Sutures placed distal to the mucocutaneous line will increase postoperative discomfort, causing induration and edema not conducive to optimal healing, and should not be used. Divulsion of the sphincters is generally disapproved. Merar[125] considers this a major factor in producing severe postoperative pain, stating that the idea of causing temporary paralysis by stretching of the sphincters is based on a false premise. Unless the muscle is actually damaged, paralysis lasts less than thirty minutes in most cases, and in the process of recovering its contractile power the muscle contrast spasmodically, causing sudden violent pain. I have for some years utilized the principles of Buie's[28] "average type" and "amputative type" methods of hemorrhoidectomy which may be adapted to the particular condition in each particular patient for suitable plastic procedure.

Injection Treatment of Hemorrhoids.—This method of treatment, originated in 1866 by Blackwood,[7] exploited by charlatans and ignorant practitioners, was for many years in disrepute because of the many unfortunate complications incurred. Today, with proper recognition of its limitations, it is a frequently used and valuable method of treatment in selected cases. It is almost universally conceded and emphasized that injection therapy is restricted to the treatment of internal hemorrhoids and should never be used for external hemorrhoids. Injection of a sclerosing solution below the mucocutaneous line produces severe pain and is usually followed by extensive painful necrosis and sloughing of tissue. Perhaps not sufficient stress has been laid upon its contraindications in the treatment of internal hemorrhoids complicated by inflammation, thrombosis, marked ulceration, or strangulation, or in the presence of associated pathologic conditions, such as cryptitis, fissure, or fistula. This method is ideally suited to the treatment of uncomplicated, superficially eroded internal hemorrhoids in the patient whose only symptom is bleeding. It may be employed for some measure of palliative relief in patients with large combined externointernal hemorrhoids who are unable for one reason or another to undergo hemorrhoidectomy. Any patient who has pain as a primary complaint must of necessity have some pathologic condition at or below the level of the mucocutaneous line and will not be relieved of his pain by injection treatment. All too frequently this error is made. Although the majority of published reports claim permanent cure in better than 85 per cent of cases, there are many proctologists today who sincerely believe that this form of treatment exceptionally produces permanent cure. When treatment is advised

and undertaken, their patients are told that its effects may not be expected to be permanent, but that it will satisfactorily relieve their symptoms for a period of from one to five years and that further treatment may eventually be required. Some of this group believe that treatment should be continued only to the point of relief of symptoms, usually bleeding, rather than to attempt by repeated treatments to entirely eliminate all traces of internal hemorrhoids.

The technique of injection consists in depositing a sclerosing solution through the mucous membrane into the submucosa containing the hemorrhoidal veins. A chemical inflammatory reaction results with influx of leucocytes and the ultimate production of fibrous tissue which in its contraction compresses and obliterates the hemorrhoidal veins. It has been recommended that injection be done just at the upper limits of the internal hemorrhoid, thereby obliterating the superior hemorrhoidal veins above the varicosities, with the result that subsequently the remainder of the hemorrhoid will be smaller in size and more readily sclerosed. Also such high injection is suggested to fix the upper extent of the hemorrhoid so that when subsequent injection is done into the main mass of the hemorrhoid and temporary swelling occurs there will be no danger of prolapse and incarceration. The interval between treatments varies from five to fourteen days. Commonly, treatment is given once weekly, the same hemorrhoid not being injected oftener than every second week. The number of treatments given varies from one to fourteen, the average being six to eight. The amount of solution injected varies for different solutions used, but there also is a difference of opinion as to the relative amount of solution that should be injected. Some employ from a few minims to 1 c.c. Others distend the hemorrhoid until all folds are obliterated and the mucosa shows definite pallor. Ordinarily pallor of the mucous membrane is an indication that the injection is being made too superficially and necrosis is liable to ensue if injection is continued. Competent treatment by injection necessitates thorough knowledge of the anatomy of the anorectum and a certain adeptness in the development of proper exposure with a suitable instrument.

It is impossible to estimate in what percentage of patients injection treatment is to be recommended. Essentially all proctologists employ this method in at least some patients. The number of patients who are given palliative treatment by this means in order to avoid loss of time and avoidance of expense swells the proportion who are so treated probably to about 50 per cent. It is only the rare misguided enthusiast who has abandoned operative treatment entirely for injection therapy.[19] In general the extensive enthusiasm of a decade ago has been tempered by experience and this method of treatment is now being given its more proper position of importance in the treatment of hemorrhoids.

Sclerosing Solutions.—The ideal reagent for injection treatment is one which will produce sufficient irritation and reaction to result in adequate fibrosis without actually producing necrosis. Absence of toxicity and idiosyncrasy is desirable. Many agents have been used, including 70 per cent alcohol,[20] epinephrine, hydrochloric acid, ferric chloride, 50 per cent glucose,[123, 124] hypertonic sodium chloride, horse serum, salicylic acid, trinitrophenol, quinine 30 per cent solution,[19] quinine and urethane, quinine and urea dichlorhydrolactate, sodium morrhuate and zinc chloride. At the present time, however, there are only two reagents widely used for the purpose, quinine and urea hydrochloride and phenol in vegetable oil.

Quinine and urea hydrochloride (5 per cent) was originally suggested by Terrell[181] and has been found very satisfactory by many.[9, 11, 12, 28, 37, 97, 105, 156, 163, 168] As a rule from 0.5 to 1 c.c. is injected into each hemorrhoid. Although idiosyncrasy is rare, a careful history should be ascertained before treatment. Sloughing occurs only as the result of faulty technique due to such errors as too superficial injection, injection too close to the mucocutaneous line, injecting too much solution or a second injection into the same hemorrhoid after too short an interval of time.

Phenol has long been a popular reagent for injection of internal hemorrhoids. When originally used in as high as 20 per cent and 30 per cent solutions, necrosis and sloughing occurred in a fairly high percentage of cases, so that a certain prejudice remains today to its use. However, 5 per cent phenol in vegetable oil has been found to produce about the same degree of reaction as 5 per cent quinine and urea hydrochloride[7] and is used by a considerable number of proctologists.[43, 166, 189, 195] Formerly glycerin was used as a vehicle for phenol, but chemical stricture from the continued reaction to the unabsorbed glycerin has been reported.[153] It has been found that this objection is corrected by the use of vegetable oil in place of glycerin. Five per cent phenol in vegetable oil is being used in amounts of from 1 to 10 c.c. injected into a single hemorrhoid by the same technique as that used for quinine and urea hydrochloride. The amount to be injected may be determined by the degree of redundancy of the mucous membrane that is present.[43] As with quinine and urea hydrochloride, sloughing should not occur if proper technique is employed.

Electrical Treatment of Hemorrhoids.—Occasional reports are found in the literature extolling the advantages of positive or negative galvanism, "coagulation" by bipolar diathermy, or "desiccation" by monopolar high frequency current.[3, 17, 21, 64, 65, 68, 80, 116, 162, 184] This subject can be adequately covered by two quotations:

1. Buie:[28] "The so-called coagulating needle has been in use for several decades and is widely used by those who claim to have certain

secret methods which are painless and which 'dissolve' the hemorrhoid instead of cutting it out."

2. Kleckner:[105] "This method . . . requires expensive equipment and thorough training to accomplish in a round-about way what can be done more easily, more quickly and equally as well by such certain methods as injection and operation."

PROLAPSE

Classification.—For many years protrusion of the rectal mucosa through the anus has been called incomplete or partial prolapse and invagination of all the coats of the rectal wall has been called complete prolapse. The latter has been subdivided into three types: first degree (external displacement of the lower rectal wall with complete eversion of the anal canal), second degree (external displacement of the lower rectal wall without disturbance of the wall of the anal canal), and third degree (invagination of the upper rectal or sigmoid wall into the rectal ampulla without external displacement). Martin[119] in 1932 argued that this classification should be reversed. Buie[28] divides the classification into two chief headings. "Visible prolapse" is subdivided into the partial type (incomplete prolapse) and the complete type, first degree and second degree (comparable in old classification). "Concealed rectal prolapse" is comparable to third degree complete prolapse in the old classification. Bacon,[8] following Martin,[118] proposes a change in nomenclature, using the term "prolapse" to apply only to the incomplete or partial type and utilizing the term "procidentia" for abnormal descent of all coats of the rectum.

Treatment.—The many operative procedures that have been advocated for the treatment of rectal prolapse are mute evidence of the fact that such procedures are all too often followed by disappointment and recurrence. The principle methods of rectorrhaphy, rectopexy, colopexy, and amputative resection are well described by Bacon.[7] Buie[28] describes an amputative resection which he believes, properly performed, will correct far more of these conditions than have been previously accredited to it, even to the most extensive prolapse. C. W. Mayo[120] recently described a new intra-abdominal technique in which multiple fascial strips are utilized for obliteration and fixation of the median perineal hernia. He considers this an important correction of a major weakness in the classical operation of Moscowitz.[133] Murrieta[138] has reported recently a unique operation applicable to all forms and degrees of prolapse and procidentia based upon his technique of valvotomy.[139] The use of the elastic ligature method of amputation has been largely discontinued although articles recommending its use in selected cases have appeared by Reid, Wangensteen, and Eliason.

Great advances in the treatment of prolapse have been made by the use of injections of sclerosing solutions to produce fibrosis and support. This method has been found to be almost uniformly successful in all degrees of incomplete prolapse and frequently has been efficacious in the correction of variable degrees of complete prolapse. Many now believe that all degrees of prolapse should be treated by injection and only if failure results should operation be considered. Smith[165] has not found it necessary to do a surgical operation, other than the injection of hydrochloric acid, in ten years. Hanes[74] claims that he has never failed to cure a patient who didn't have straining. Both submucosa and perirectal injection may be utilized.

Hydrochloric Acid.—Hanes[75, 76] injects 50 to 60 c.c. of 1:3,000 hydrochloric acid into the perirectal tissues, preceded by a similar amount of 0.5 per cent novocaine. Smith[166] uses a dilution of 1:1,500. I have obtained very satisfactory results by the use of the latter dilution in amounts up to 240 c.c. injected with the patient under spinal anesthesia without local novocaine infiltration.

Quinine and Urea Hydrochloride.—Terrell[179] has for fifteen years used this solution with invariable success in children, injecting 0.5 c.c. of 2 to 3 per cent solution in 2, 3, or 4 quadrants into the submucosa just above the anorectal line. Only a few cases require more than one treatment. Buie[28] uses this solution in 5 per cent strength, injecting 2 to 3 c.c. into the submucosa in 2 to 3 different places and repeating the treatment in several days. He has used the same solution for perirectal injection in mild cases of complete prolapse.

Phenol in Vegetable Oil.—This solution in 5 per cent strength has been advocated by Gabriel,[56] who makes a series of high submucosal injections, 2 c.c. in each of six to eight different sites. In combination with this he injects perirectally, posteriorly and on either side, 3 c.c. of Swinford Edwards solution (quinine sulfate gr. 12; dilute sulfuric acid, minims 30; and distilled water, minims 30, sterilized by boiling). The Gabriel technique has been followed and praised by others.[23, 42, 132] Alcohol, 70 to 95 per cent, has been advocated by many.[40, 51, 58, 127, 135, 144]

(To be continued in the July issue. The references will accompany the second section.)

Book Reviews

Textbook of Neuroanatomy and the Sense Organs. By O. Larsell, Professor of Anatomy, University of Oregon Medical School. Pp. 343, with 232 illustrations. New York, London, 1939. D. Appleton-Century Company. $6.

This meritorius book has been written primarily for first-year medical students, but it will be found extremely useful to all who wish a clear, concise treatise on the nervous system from the functional point of view.

After a brief consideration of the phylogenetic emergence of the synaptic nervous system, establishment of the reflex arc, embryology, and hystogenesis, there follows a terse description of the structure and the functional components of the peripheral nerves with clinical interpretation of some of the commoner lesions of spinal nerves. Sensory and motor endings are satisfactorily covered and well illustrated.

In the chapter on the spinal cord and its meninges, the fiber tracts are effectively treated by colored cross sections and black-and-white diagrams of the general course and connections to higher levels. One may question the relative size of some of the tracts. Chordotomy indicates that a larger area is occupied by the lateral spinothalamic tract than is allotted, particularly in the cervical cord, while the anterior (ventral) spinocerebellar tract is oversized. Technically it would seem better to place the anterior corticospinal (direct pyramidal) tract nearest the ventral median fissure throughout the cervical and upper thoracic levels instead of lateral to the sulcomarginal tract as is done here and in most texts. The ventral corticospinal tract regularly extends to the medial margin of the ventral funiculus. Clinical applications are mentioned at many points. On page 80 it is stated that in destruction of the anterior white commissure, as in syringomyelia, there is loss of pain and temperature sensations on the *opposite* side of the body, whereas the sensory loss is usually bilateral.

The approach to the brain is through the cranial nerves, which are analyzed in a masterly fashion. The description of the medulla oblongata is clarified by means of many excellent drawings. Figs. 94, 96, and 99 are not quite typical of the human brain stem with respect to the position of the tectospinal tract, which is too far dorsal and hence unduly reduces the size of the medial longitudinal bundle. Again the anterior spinocerebellar tract has been overemphasized at the expense of the lateral spinothalamic fasciculus whose clinical significance is of greater importance.

The visceral efferent and gustatory systems are briefly but accurately presented. The vestibular and auditory systems are introduced by a review of the embryology and histology of the inner ear. Well-planned cuts show the main fiber connections. A minor omission is noted in Fig. 134 where no direct descending vestibulospinal fibers in the medial longitudinal bundle are shown, although they have been clearly established by several relatively recent studies.

The cerebellum is considered under the author's new concept that it is composed of two main divisions; namely, flocculonodular lobe (vestibular in function) and corpus cerebelli (general proprioceptive). The corpus cerebelli is further divided into anterior and posterior lobes. The diagrams showing the fiber connections are very complete and undoubtedly represent the most trustworthy in existence, since the cerebellum has been the author's primary field of investigation

for many years. Functional localization and clinical interpretations are brought into line with this morphologic concept.

The midbrain and diencephalon naturally follow in this order. The various nuclei in the thalamic regions are admirably summarized as to location, structure, and function.

After some general considerations on development and lobulation of the cerebral hemispheres, basal ganglia and general organization of the white matter, brain ventricles, choroid plexus and cerebrospinal fluid, the visual system (including the eyeball) is satisfactorily covered with the usual ontogenetic introduction. The olfactory apparatus is similarly treated.

The final chapters deal with the structure of the cerebral cortex (cytoarchitecture of the better known regions), the efferent system as a unit (the pyramidal, extrapyramidal and corticopontocerebellar paths) and the function of the thalamus and cerebral localization. These topics are strictly modern.

The entire book is well planned. The language is clear and uninvolved. The nomenclature is simple. It is not an anatomic atlas but rather a functional analysis of the nervous system. There is nothing on the blood supply. Much space is spared by reducing controversial topics to a minimum. A surprising amount of neurology has been condensed into twenty-four short chapters, each of which contains a selected list of references. There is a twenty-five-page index, and there are 232 figures. Needless to say, the workmanship and material are of high quality.

Life and Letters of Fielding H. Garrison. By Solomon R. Kagan. Cloth. Pp. 287, with 3 illustrations. Boston, 1938. The Medico-Historical Press. $3.

This, the first of the biographies of America's greatest medical historian, probably will not be the last. Kagan's treatise is written in two parts. The one contains an interesting collection (selected) of Garrison's letters which reveal the warmth of his friendship for his few intimate friends and the many-sided nature of his personality. The biographical sketch of Part I is a somewhat disjointed account written under the following captions: (1) General Survey of Garrison's Life, (2) Medical Historian and Medical Bibliographer, (3) Medical Biography, (4) The Teacher, (5) Love of Music, (6) The Man, and (7) Tributes.

The volume includes a complete chronological list of Garrison's published books and articles and concludes with an appendix of varied material.

Garrison gained the admiration of his peers for his lasting achievement in the writing of a great one-volume work on medical history, which its author described under the modest title of *An Introduction to the Study of Medical History*. It will be a long time probably before this readable volume crammed with solid information unearthed by its scholarly author through years of serious and patient study will be set aside because of the advent of a superior text.

Garrison exhibited in his *Medical History* an extraordinary capacity for coordinating facts and incidents—an ability which has not fallen upon his first biographer in the same liberal measure. There is, however, much to be said for the monograph and everyone who admired Garrison and his work will want to possess this volume and gain a more intimate acquaintance with this shy and modest but talented genius. Identified as he was over a period of years with the Army Medical Library and its interests and activities, it is somewhat disconcerting to learn that he never enjoyed other than temporarily the position of Librarian of the Army Medical Library.

Diseases of the Thyroid, Parathyroid and Thymus. By Andre Crotti, M.D., formerly First Assistant of the Pathological Laboratory at University of Lausanne, Switzerland. Cloth. Ed. 3. Pp. 1229, with 262 illustrations and 39 plates in color. Philadelphia, 1938, Lea & Febiger. $20.

This encyclopedic volume contains 51 chapters and more than 50 pages of bibliography. It is essentially a clinical treatise dealing with the problems of the thyroid, parathyroids, and thymus.

The author believes in the infectious origin of goiter, although he concedes an important role to iodine. If local environment or other factors did not play a part in the pathogenesis of goiter, in other words, if goiter were a pure deficiency disease, it should be largely the same, the world over, which, of course, it is not. The author cites some experimental evidence, not very convincing, adduced by himself which purports to give the infection factor greater credence.

The author believes that the presence of a goiter sensitizes a patient to iodine. He believes also in so-called "iodin Basedow" described by Rilliet, Breuer, and Kocher, a thesis which this reviewer believes is still demanding of proof. The author does not believe that factual data warrant drawing a sharp distinction between exophthalmic goiter and adenomatous goiter with hyperthyroidism, an attitude which the reviewer shares. The author speaks of hyperthyroidism as a "thyro-neuro-polyglandular disease."

Crotti's book constitutes a valuable source book containing a great mass of factual data interspersed between which lie many impressionistic ideas of the author; whether the latter add to the value of the volume or detract from its worth, it must be conceded, is a debatable issue.

The Surgical Treatment of Hypertension. By George Crile. Pp. 239, with 52 illustrations. Philadelphia, 1938, W. B. Saunders Company. $4.

The monograph, *The Surgical Treatment of Hypertension* by Dr. George Crile, describes the personal point of view of its author on the etiology of essential hypertension and its attack by resection of the celiac ganglia. Crile has formulated the theory that since energy output of the organism is controlled by the brain, thyroid, and sympathoadrenal system, and since this triad is most highly developed in the lion and in man, the cardiovascular systems of these species are subjected to the greatest strain. His report that the large cats develop arteriosclerosis when kept in captivity is of great interest. Whereas the lion, once his belly is filled, lies down to sleep, man is in a perpetual state of activity. The sequel to this constant drive is the induction of scarring in the arterial tree and the development of hypertension. In further support of this hypothesis Crile states that the disease is due to an inherited large size of the celiac ganglia and the associated nerve structures which he has observed at operation. As listed by the author, the most important links in this system of energy control are the adrenal medulla and the celiac and aortic plexuses. Little mention is made of all the recent work on the autonomic pressor centers in the premotor cortex and diencephalon, but the medulla of the adrenal gland is considered as the "brain" of the sympathetic system and the genesis of human hypertension ascribed to "a pathologic physiology" of the celiac, aortic, and adrenal nerve plexuses. Few clinicians will subscribe to the author's statement that "hyperthyroidism, neurocirculatory asthenia, and Raynaud's disease are abated or cured by adrenal denervation or celiac ganglionectomy."

The ideas above are stimulating and provocative to a new line of thought, but as one reads the book through one finds numerous exaggerations, such as that

the charging lion travels at the rate of a mile a minute, something that no conservative naturalist will substantiate. There is also so much neglect of accepted scientific fact that one's credulity is further strained. For instance, no such vein as the "celiac" is listed in Gray's *Anatomy*. Physiologists who have had experience with denervating the adrenal in the dog and cat know that all its nerve fibers do not come through the celiac ganglion, as many accessory ones reach it from the upper portion of the lumbar sympathetic chain. While this point has not been definitely settled in man, the assumption that celiac ganglionectomy results in complete denervation of the adrenal is not justified. Furthermore, granted that the celiac ganglia contain the nerve cells of all the postganglionic fibers to the upper abdominal viscera, their destruction must be followed by such an increased sensitivity to adrenaline and sympathin that vasoconstriction of the vascular bed cannot be greatly reduced.

The operation which Crile advocates to lessen the drive on the cardiovascular system consists of excision of the celiac ganglia and denervation of the aorta and adrenal glands. This complex network of ganglia and delicate nerve fibers is identified by palpation with the hand inserted to the wrist in the retroperitoneal space under the twelfth rib. By "touch anatomy" the operator must identify the major splanchnic nerve as it emerges from the crus of the diaphragm, the adrenal gland, as well as the surrounding arteries and veins. The celiac ganglion must then be freed up and its branches cut away from the surrounding vessels by a long scissors inserted along the tip of the index finger. The major splanchnic nerve "is the only leading string to the most important unit in this anatomical jungle." The surgeon must differentiate fine nerve strands from small veins and particularly avoid injury to the blood supply of the adrenal or deep hemorrhage. While such a dissection is possible for the author, who has denervated countless adrenals for neurocirculatory asthenia, hyperthyroidism, peptic ulcer and Raynaud's disease, one must protest against its adoption by the ordinary surgeon. Even if the operator avoids the complications mentioned above, it must be borne in mind that it is an established physiologic fact that any sympathetic fibers which are left behind can maintain the original tone of the partly denervated structures. Inasmuch as at least two satisfactory approaches have been used to expose this entire area to direct visual dissection, why should it ever be attempted without the help of the eyes?

An examination of the follow-up statistics presented will fail to convince the critical clinician that the patients who have been submitted to celiac ganglionectomy have achieved as significant a reduction in blood pressure as has been observed in other clinics after intrathoracic excision of the splanchnic nerves, or after a subdiaphragmatic resection which includes the upper portion of the lumbar sympathetic trunks as well as the structures removed by Crile. Figures given in Table 9 show an average reduction in 85 cases from 217/133 to 169/110 before discharge from the hospital. One year postoperatively 16 patients are recorded as having pressures averaging 196/121. There was striking improvement in the subjective symptoms of many of these patients, but this has followed other operations for this condition. Very little excuse can be seen for advocating an incomplete method of surgical denervation by a blind approach, when far better operative exposures of these structures are in use in other clinics.

The whole status of sympathectomy in the treatment of hypertension is still definitely on trial. Surgeons have received little encouragement from animal investigation and the leading medical authorities who have studied the disease are still not convinced that real benefit follows sympathectomy. A careful perusal of this book has not convinced the reviewer that it has advanced our understanding of essential hypertension.

Medicine in Modern Society. By David Riesman. Cloth. Pp. 226. Princeton, N. J., 1938, Princeton University Press. $2.50.

The author of this book is a widely known, admired, and beloved clinician, teacher, scholar, and investigator. Dr. Riesman's many papers in the fields of pathology, medicine, and medical history have been so greatly appreciated by medical readers that they welcome enthusiastically the announcement of this new semihistorical, semiphilosophical book from his pen.

Medicine in Modern Society was written for the general rather than for the medical reader; but physicians will find that familiar facts are here presented in a new and interesting setting and that the controversial subject of the dispensation of medical care is discussed frankly and realistically.

The basis of this book was a series of Princeton University lectures, the Vanuxem Lectures, in the development of which the author was guided by his belief that "the history of medicine is in reality an epitome of the history of civilization and should form a part of every man's culture."

In a simple, fascinating manner Dr. Riesman paints a picture of the high points in the development of the science and the art of medicine. Hippocrates, Galen, Vesalius—"the Columbus of anatomy"—Harvey, Jenner, McDowell, Long, Morton, Simpson, Semmelweis, Holmes, Lister, Pasteur, Koch and others down to the present day all take their places briefly but impressively in the pageant of medical history which passes before the reader in a few brief pages.

Opium, mercury, cinchona, digitalis, vaccination, anesthesia, antisepsis, antitoxins, x-ray, radium, salvarsan, insulin, liver therapy, allergy, endocrinology, psychoanalysis, hormones, vitamines, viruses, and even fever therapy and sulfanilamide are briefly and deftly fitted into the picture. Many well-chosen anecdotes and personal experiences add interest to the story.

The methods and the problems involved in arriving at a diagnosis and in deciding upon a course of treatment are presented in a way that should make for better understanding between patient and doctor. In discussing the importance of an accurate history of the patient's illness and the delight that some patients seem to take in refusing to answer the physician's questions truthfully or completely, he recalls "a story told of the great Iron Chancellor. He had had many physicians, all of whom he discharged. Finally he summoned a Doctor Schweninger, who began to ask him many questions. Impatiently Bismarck said to the doctor, 'You should not ask so much—you should know what ails me without questioning.' 'Your Highness,' answered the doctor, 'I would suggest you call a veterinarian; he asks no questions.' "

The present status of medicine and medical education is described as exemplifying the best of our civilization. Medical cults and superstitions are explained. The roles of the specialist and the family physician of the future are set forth so clearly and so sanely that everyone can understand.

The chapter on the social outlook of medicine is a thoughtful, realistic, and sane consideration of the social problems today which the medical profession is confronted with. That certain problems exist Dr. Riesman admits, but he concludes that "a profession that has elevated the standards of medical education to a level unequalled in the civilized world, has regulated hospitals until today no nation can match ours, publishes the best medical journal in existence together with many other works that serve to educate the American physician; brings together annually thousands of doctors from all over the land for a week of intensive postgraduate study which is a model for the world—such an Association can I think be trusted to collaborate with socially minded laymen toward a plan for distributing medical care, both curative and preventive, to all people." On the other hand, he says, this Association "must take the lead in the inevitable movement of reform. If it fails to lead, then it will be obliged to follow those who have neither the knowledge, the wisdom, nor the incentive to preserve what is best in American medicine."